```
=> fil reg
FILE 'REGISTRY' ENTERED AT 16:12:26 ON 23 MAR 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2006 American Chemical Society (ACS)

Property values tagged with IC are from the ZIC/VINITI data file
provided by InfoChem.

STRUCTURE FILE UPDATES:   22 MAR 2006  HIGHEST RN 877759-05-2
DICTIONARY FILE UPDATES:  22 MAR 2006  HIGHEST RN 877759-05-2

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH January 6, 2006

  Please note that search-term pricing does apply when
  conducting SmartSELECT searches.

*********************************************************************
*                                                                   *
* The CA roles and document type information have been removed from *
* the IDE default display format and the ED field has been added,   *
* effective March 20, 2005.  A new display format, IDERL, is now    *
* available and contains the CA role and document type information. *
*                                                                   *
*********************************************************************

Structure search iteration limits have been increased.  See HELP SLIMITS
for details.

REGISTRY includes numerically searchable data for experimental and
predicted properties as well as tags indicating availability of
experimental property data in the original document.  For information
on property searching in REGISTRY, refer to:

http://www.cas.org/ONLINE/UG/regprops.html

=> d l14 sqide can

L14  ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2006 ACS on STN
RN   83139-29-1  REGISTRY
CN   L-Phenylalaninamide, L-seryl-L-valyl-L-seryl-L-α-glutamyl-L-
     isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-asparaginyl-L-
     leucylglycyl-L-lysyl-L-histidyl-L-leucyl-L-asparaginyl-L-seryl-L-methionyl-
     L-α-glutamyl-L-arginyl-L-valyl-L-α-glutamyl-L-tryptophyl-L-
     leucyl-L-arginyl-L-lysyl-L-lysyl-L-leucyl-L-glutaminyl-L-α-aspartyl-
     L-valyl-L-histidyl-L-asparaginyl- (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   1-34-Human PTH amide
CN   1-34-Human PTH-NH2
CN   HPTH-(1-34)-NH2
CN   Human parathyroid hormone(1-34) amide
FS   PROTEIN SEQUENCE
SQL  34

SEQ      1 SVSEIQLMHN LGKHLNSMER VEWLRKKLQD VHNF
           ========== ========== ========== ====
HITS AT:   1-34
```

RELATED SEQUENCES AVAILABLE WITH SEQLINK
DR 120945-80-4
MF C181 H292 N56 O50 S2
CI MAN
LC STN Files: CA, CAPLUS, CASREACT, CHEMCATS, TOXCENTER, USPATFULL
DT.CA CAplus document type: Conference; Journal; Patent
RL.P Roles from patents: BIOL (Biological study); PREP (Preparation); RACT (Reactant or reagent); USES (Uses)
RL.NP Roles from non-patents: BIOL (Biological study); PREP (Preparation); PROC (Process); PRP (Properties); USES (Uses)
RLD.NP Roles for non-specific derivatives from non-patents: BIOL (Biological study); PRP (Properties); USES (Uses)
31 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
31 REFERENCES IN FILE CAPLUS (1907 TO DATE)

REFERENCE 1: 143:472988

REFERENCE 2: 142:274210

REFERENCE 3: 141:17797

REFERENCE 4: 141:1641

REFERENCE 5: 140:164240

REFERENCE 6: 139:391567

REFERENCE 7: 139:375612

REFERENCE 8: 138:331836

REFERENCE 9: 135:10001

REFERENCE 10: 134:305606

=> fil hcaplus
FILE 'HCAPLUS' ENTERED AT 16:12:34 ON 23 MAR 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

FILE COVERS 1907 - 23 Mar 2006 VOL 144 ISS 13
FILE LAST UPDATED: 22 Mar 2006 (20060322/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate

substance identification.

=> d l18 bib abs hitrn retable tot

L18 ANSWER 1 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 2005:1221085 HCAPLUS
DN 143:472988
TI Method for fostering bone formation and preservation
IN Vignery, Agnes; **Mehta, Nozer M.; Gilligan, James P.**
PA USA
SO U.S. Pat. Appl. Publ., 19 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005256047	A1	20051117	US 2005-128095	20050511
WO 2005112864	A2	20051201	WO 2005-US16613	20050512

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2004-571200P	P	20040514	
US 2005-128095	A	20050511	

AB A method of inducing bone formation in a subject in need of such inducement comprises the steps of mech. inducing an increase in osteoblast activity in the subject and elevating blood concentration of at least one bone anabolic agent in the subject. The method steps may be performed in any order, but in sufficient time proximity that the elevated concentration of the anabolic agent and the mech. induced increase in osteoblast activity overlaps. The method may addnl. comprise providing the subject with an elevated blood concentration of at least one antiresorptive agent, wherein the elevated concentration is sufficient to prevent resorption of new bone growth produced due to the osteoblast activity. Use of the method permits targeting of specific bones of the subject for bone production and preservation, faster bone production and earlier discontinuation of bone anabolic pharmaceuticals. Kits adapted for performing the method are provided.
IT **83139-29-1**
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(method for fostering bone formation and preservation using anabolic agent to increase osteoblast activity and antiresorptive agent)

L18 ANSWER 2 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 2005:184232 HCAPLUS
DN 142:274210
TI Bioactive N-terminal undecapeptides derived from parathyroid hormone: the role of α-helicity
AU Barazza, A.; Wittelsberger, A.; Fiori, N.; Schievano, E.; Mammi, S.; Toniolo, C.; Alexander, J. M.; Rosenblatt, M.; Peggion, E.; Chorev, M.

CS Division of Bone and Mineral Research, Beth Israel Deaconess Medical Center and Harvard Medical School, Boston, MA, 02215, USA
SO Journal of Peptide Research (2005), 65(1), 23-35
CODEN: JPERFA; ISSN: 1397-002X
PB Blackwell Publishing Ltd.
DT Journal
LA English
AB The N-terminal 1-34 segment of parathyroid hormone (PTH) is fully active in vitro and in vivo and it can reproduce all biol. responses in bone characteristic of the native intact PTH. Recent studies have demonstrated that N-terminal fragments presenting the principal activating domain such as PTH(1-11) and PTH(1-14) with helicity-enhancing substitutions yield potent analogs with PTH(1-34)-like activity. To further investigate the role of α-helicity on biol. potency, we designed and synthesized by solid-phase methodol. the following hPTH(1-11) analogs substituted at positions 1 and/or 3 by the sterically hindered and helix-promoting Cα-tetrasubstituted α-amino acids α-amino isobutyric acid (Aib), 1-aminocyclopentane-1-carboxylic acid (Ac5c) and 1-aminocyclohexane-1-carboxylic acid (Ac6c): Ac5c-V-Aib-E-I-Q-L-M-H-Q-R-NH2 (I); Aib-V-Ac5c-E-I-Q-L-M-H-Q-R-NH2 (II); Ac6c-V-Aib-E-I-Q-L-M-H-Q-R-NH2 (III); Aib-V-Ac6c-E-I-Q-L-M-H-Q-R-NH2 (IV); Aib-V-Aib-E-I-Q-L-M-H-Q-R-NH2 (V); S-V-Aib-E-I-Q-L-M-H-Q-R-NH2 (VI), S-V-Ac5c-E-I-Q-L-M-H-Q-R-NH2 (VII); Ac5c-V-S-E-I-Q-L-M-H-Q-R-NH2 (VIII); Ac6c-V-S-E-I-Q-L-M-H-Q-R-NH2 (IX); Ac5c-V-Ac5c-E-I-Q-L-M-H-Q-R-NH2 (X); Ac6c-V-Ac6c-E-I-Q-L-M-H-Q-R-NH2 (XI). All analogs were biol. evaluated and conformationally characterized in 2,2,2-trifluoroethanol (TFE) solution by CD. Analogs I-V, which cover the full range of biol. activity observed in the present study, were further conformationally characterized in detail by NMR and computer simulations studies. The results of ligand-stimulated cAMP accumulation expts. indicated that analogs I and II are active, analogs III, VI and VII are very weakly active and analogs IV, V, VIII-XI are inactive. The most potent analog, I exhibits biol. activity 3500-fold higher than that of the native PTH(1-11) and only 15-fold weaker than that of the native sequence hPTH(1-34). Remarkably, the two most potent analogs, I and II, and the very weakly active analogs, VI and VII, exhibit similar helix contents. These results indicate that the presence of a stable N-terminal helical sequence is an important but not sufficient condition for biol. activity.
IT **83139-29-1**, HPTH(1-34)NH2
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(bioactive N-terminal undecapeptides derived from parathyroid hormone and role of α-helicity)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Arad, O	1990	3	42	Pept Res	HCAPLUS
Barazza, A	2004		673	Peptide Revolution:	
Barden, J	1996	220	431	Biochem Biophys Res	HCAPLUS
Barden, J	1993	32	7126	Biochemistry	HCAPLUS
Barden, J	1989	184	379	Eur J Biochem	HCAPLUS
Bardi, R	1986	25	1635	Biopolymers	HCAPLUS
Bax, A	1985	65	355	J Magn Reson	HCAPLUS
Bull, T	1988	80	470	J Magn Reson	
Bundi, A	1978	91	201	Eur J Biochem	HCAPLUS
Bundi, A	1976	64	126	FEBS Lett	HCAPLUS
Carpino, L	1994	2	203	J Chem Soc, Chem Com	
Chen, Z	2000	39	12766	Biochemistry	HCAPLUS
Chorev, M	2002		423	Principles of Bone B	HCAPLUS
Chorev, M	2001		53	The Parathyroids --	HCAPLUS

Gisin, B	1972	58	248	Anal Chim Acta	HCAPLUS
Greenfield, N	1969	8	4108	Biochemistry	HCAPLUS
Griesinger, C	1988	110	7870	J Am Chem Soc	HCAPLUS
Gronwald, W	1997	378	1501	Biol Chem Hoppe-Seyl	HCAPLUS
Jin, L	2000	275	27238	J Biol Chem	HCAPLUS
Kaiser, E	1970	34	595	Anal Biochem	HCAPLUS
Kanaori, K	1997	249	878	Eur J Biochem	HCAPLUS
Karle, I	1990	29	6747	Biochemistry	HCAPLUS
Kaul, R	1999	7	105	Bioorg Med Chem	HCAPLUS
Klaus, W	1991	30	6936	Biochemistry	HCAPLUS
Lee, E	2004	45	419	Yonsei Med J	HCAPLUS
Lee, S	1989	28	1115	Biopolymers	HCAPLUS
Lim, S	2004	64	25	J Pept Res	HCAPLUS
Marx, U	2000	267	213	Biochem Biophys Res	HCAPLUS
Marx, U	1995	270	15194	J Biol Chem	HCAPLUS
Marx, U	1998	273	4308	J Biol Chem	HCAPLUS
Merrifield, R	1963	85	2149	J Am Chem Soc	HCAPLUS
Nakamoto, C	1995	34	10546	Biochemistry	HCAPLUS
Neer, R	2001	344	1434	N Engl J Med	HCAPLUS
Neugebauer, W	1995	34	8835	Biochemistry	HCAPLUS
Pastore, A	1990	90	165	J Magn Reson	HCAPLUS
Paul, P	1986	108	6363	J Am Chem Soc	HCAPLUS
Pavone, V	1988	21	2064	Macromolecules	HCAPLUS
Pellegrini, M	1998	37	12737	Biochemistry	HCAPLUS
Pellegrini, M	1998	273	10420	J Biol Chem	HCAPLUS
Pines, M	1994	135	1713	Endocrinology	HCAPLUS
Rance, M	1983	117	479	Biochem Biophys Res	HCAPLUS
Ray, F	1993	211	205	Eur J Biochem	HCAPLUS
Santini, A	1988	10	292	Int J Biol Macromol	HCAPLUS
Schievano, E	2000	54	429	Biopolymers	HCAPLUS
Shimizu, M	2001	142	3068	Endocrinology	HCAPLUS
Shimizu, M	2000	275	21836	J Biol Chem	HCAPLUS
Shimizu, N	2003	42	2282	Biochemistry	HCAPLUS
Shimizu, N	2001	276	49003	J Biol Chem	HCAPLUS
Smith, L	1987	253	81	Arch Biochem Biophys	HCAPLUS
Strickland, L	1993	32	6050	Biochemistry	HCAPLUS
Toniolo, C	2001	60	396	Biopolymers (Pept Sc	HCAPLUS
Toniolo, C	1991	16	350	Trends Biochem Sci	HCAPLUS
Tsomaia, N	2004	43	690	Biochemistry	HCAPLUS
Valle, G	1988	II	393	J Chem Soc, Perkins	
Weidler, M	1999	444	239	FEBS Lett	HCAPLUS
Wittelsberger, A	2004		679	Peptide Revolution:	
Wray, V	1994	33	1684	Biochemistry	HCAPLUS

L18 ANSWER 3 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 2004:429384 HCAPLUS
DN 141:17797
TI Ligand-selective dissociation of activation and internalization of the parathyroid hormone (PTH) receptor: conditional efficacy of PTH peptide fragments
AU Sneddon, W. Bruce; Magyar, Clara E.; Willick, Gordon E.; Syme, Colin A.; Galbiati, Ferruccio; Bisello, Alessandro; Friedman, Peter A.
CS Department of Pharmacology, University of Pittsburgh School of Medicine, Pittsburgh, PA, 15261, USA
SO Endocrinology (2004), 145(6), 2815-2823
CODEN: ENDOAO; ISSN: 0013-7227
PB Endocrine Society
DT Journal
LA English
AB G protein-coupled receptors (GPCRs) mediate the action of many hormones,

cytokines, and sensory and chemical signals. It is generally thought that receptor desensitization and internalization require occupancy and activation of the GPCR. PTH and PTHrP receptor (PTH1R) belongs to GPCR class B and is the major regulator of extracellular calcium homeostasis. Using kidney distal convoluted tubule cells transfected with a human PTH1R/enhanced green fluorescent protein fusion protein, quant., real-time fluorescence microscopy was used to analyze receptor internalization. In these cells, which are the target of the calcium-sparing action of PTH, PTH(1-34) activated adenylyl cyclase (AC) and phospholipase C (PLC) and PTH1R endocytosis. PTH(1-31), however, stimulated AC and PLC but not PTH1R endocytosis. Conversely, PTH(7-34) rapidly stimulated PTH1R internalization without activating AC or PLC. PTH(2-34) and (3-34) caused PTH1R internalization intermediate between PTH(1-34) and (7-34). PTH1R sequestration occurred in a dynamin- and clathrin-dependent manner. Directly activating AC inhibited PTH1R internalization in response to PTH(7-34). PTH1R endocytosis was sensitive to protein kinase C inhibition. PTH(1-34), (7-34), and (1-31) evoked PTH1R phosphorylation. Removal of most of the C terminus of the PTH1R eliminated receptor phosphorylation and the cAMP/protein kinase C sensitivity of internalization. PTH(1-34) and (7-34) internalized the truncated PTH1R with identical kinetics, and the response was unaffected by forskolin. Thus, the PTH1R C terminus contains regulatory sequences that are involved in, but not required for, PTH1R internalization. The results demonstrate that receptor activation and internalization can be selectively dissociated

IT **83139-29-1**, Human PTH 1-34 amide
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(PTH peptide fragments-selective dissociation of activation and internalization of parathyroid hormone (PTH) receptor)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Anborgh, P	2000	14	2040	Mol Endocrinol	HCAPLUS
Bacskai, B	1990	347	388	Nature	HCAPLUS
Barbier, J	1997	40	1373	J Med Chem	HCAPLUS
Bisello, A	2002	277	38524	J Biol Chem	HCAPLUS
Blind, E	1995	136	4271	Endocrinology	HCAPLUS
Blind, E	1996	11	578	J Bone Miner Res	HCAPLUS
Carpio, L	2001	281	E489	Am J Physiol Endocri	HCAPLUS
Castro, M	2002	143	3854	Endocrinology	HCAPLUS
Claing, A	2002	66	61	Prog Neurobiol	HCAPLUS
Daaka, Y	1998	273	685	J Biol Chem	HCAPLUS
de Rooij, J	1998	396	474	Nature	HCAPLUS
Dicker, F	1999	96	5476	Proc Natl Acad Sci U	HCAPLUS
Ferguson, S	2001	53	1	Pharmacol Rev	HCAPLUS
Ferrari, S	1999	274	29968	J Biol Chem	HCAPLUS
Ferrari, S	2001	15	149	Mol Endocrinol	HCAPLUS
Friedman, P	1996	137	13	Endocrinology	HCAPLUS
Friedman, P	1999	140	301	Endocrinology	HCAPLUS
Gardella, T	1996	137	3936	Endocrinology	HCAPLUS
Gardella, T	2001	12	210	Trends Endocrinol Me	HCAPLUS
Gesek, F	1993	264	F744	Am J Physiol	HCAPLUS
Gesek, F	1992	90	749	J Clin Invest	HCAPLUS
Gether, U	2000	21	90	Endocr Rev	HCAPLUS
Goldring, S	1985	24	513	Biochemistry	HCAPLUS
Horiuchi, N	1983	220	1053	Science	HCAPLUS
Karin, N	1999	27	681	Biotechniques	HCAPLUS
Kenakin, T	1995	16	232	Trends Pharmacol Sci	HCAPLUS
Kirchhausen, T	1999	15	705	Annu Rev Cell Dev Bi	HCAPLUS

Malecz, N	1998	12	1846	Mol Endocrinol	HCAPLUS
Morley, P	1999	6	1095	Curr Med Chem	HCAPLUS
Nabi, I	2003	161	673	J Cell Biol	HCAPLUS
Oakley, R	2001	276	19452	J Biol Chem	HCAPLUS
Orlandi, P	1998	141	905	J Cell Biol	HCAPLUS
Roth, B	1999	23	629	Neuron	HCAPLUS
Salomon, Y	1974	58	541	Anal Biochem	HCAPLUS
Sneddon, W	2000	141	4185	Endocrinology	HCAPLUS
Sneddon, W	2003	278	43787	J Biol Chem	HCAPLUS
Tawfeek, H	2002	16	1	Mol Endocrinol	HCAPLUS
Toullec, D	1991	266	15771	J Biol Chem	HCAPLUS
Tyson, D	2002	143	674	Endocrinology	HCAPLUS
Vilardaga, J	2001	276	33435	J Biol Chem	HCAPLUS
Vilardaga, J	2002	277	8121	J Biol Chem	HCAPLUS
Volonte, D	2002	13	2502	Mol Biol Cell	HCAPLUS
Walker, J	1999	274	31515	J Biol Chem	HCAPLUS

L18 ANSWER 4 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 2004:371867 HCAPLUS
DN 141:1641
TI Treatment with parathyroid hormone hPTH(1-34), hPTH(1-31), and monocyclic hPTH(1-31) enhances fracture strength and callus amount after withdrawal fracture strength and callus mechanical quality continue to increase
AU Andreassen, T. T.; Willick, G. E.; Morley, P.; Whitfield, J. F.
CS Department of Connective Tissue Biology, Institute of Anatomy, University of Aarhus, Aarhus, Den.
SO Calcified Tissue International (2004), 74(4), 351-356
CODEN: CTINDZ; ISSN: 0171-967X
PB Springer-Verlag New York Inc.
DT Journal
LA English
AB The influence of intermittent hPTH(1-34)NH2, hPTH(1-31)NH2, and monocyclic [Leu27]cyclo (Glu22-Lys26)hPTH(1-31)NH2 treatment on callus formation, mech. strength, and callus tissue mech. quality of tibial fractures in rats was investigated after 8 and 16 wk of healing. In the 8 wk of healing animals, the PTH-peptides were injected s.c. during the entire observation period (15 nmol/kg/day [hPTH(1-34)NH2: 15 nmol = 60 μg]), and control animals with fractures were given vehicle. In the 16 wk of healing animals, the PTH-peptides were injected only during the first 8 wk of healing (15 nmol/kg/day), after which the animals were left untreated during the rest of the healing period. After the first 8 wk of healing, increased fracture strength and callus volume were seen in the PTH-treated rats (ultimate load 66%, ultimate stiffness 58%, callus volume 28%), and the three peptides were equally effective. No difference in callus tissue mech. quality was found between PTH and vehicle animals. After 16 wk of healing, no differences in fracture strength, callus volume, or callus tissue mech. quality were seen between PTH and vehicle. When comparing PTH-treated animals at 8 and 16 wk, fracture strength and callus tissue mech. quality continued to increase after the withdrawal of PTH (ultimate load 23%, ultimate stress 88%, elastic modulus 87%) and external callus volume declined during this period (27%).
IT **83139-29-1**, Human PTH(1-34) amide
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(parathyroid hormone enhancement of bone fracture strength and callus amount after withdrawal)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File

Andreassen, T	2001	72	304	Acta Orthop Scand	MEDLINE
Andreassen, T	1999	14	960	J Bone Miner Res	HCAPLUS
Andreassen, T	2000	15	2266	J Bone Miner Res	HCAPLUS
Bak, B	1991	264	295	Clin Orthop	
Barbier, J	1997	40	1373	J Med Chem	HCAPLUS
Bostrom, M	2000	26	437	Bone	HCAPLUS
Dobnig, H	1995	136	3632	Endocrinology	HCAPLUS
Ejersted, C	1998	23	43	Bone	HCAPLUS
Ejersted, C	1998	62	316	Calcif Tissue Int	HCAPLUS
Ejersted, C	1994	130	201	Eur J Endocrinol	HCAPLUS
Ejersted, C	1993	8	1097	J Bone Miner Res	HCAPLUS
Fraher, L	1999	84	2739	J Clin Endocrinol Me	HCAPLUS
Holzer, G	1999	366	258	Clin Orthop	
Jahng, J	2000	23	1089	Orthopedics	MEDLINE
Jorgensen, P	1991	12	353	Bone	MEDLINE
Lindsay, R	1997	350	550	Lancet	HCAPLUS
Marcus, R	2003	18	18	J Bone Miner Res	HCAPLUS
Mohan, S	2000	27	471	Bone	HCAPLUS
Morley, P	2001	68	95	Calcif Tissue Int	HCAPLUS
Mosekilde, L	1995	16	223	Bone	HCAPLUS
Mosekilde, L	1994	134	2126	Endocrinology	HCAPLUS
Nakajima, A	2002	17	2038	J Bone Miner Res	HCAPLUS
Neer, R	2001	344	1434	N Engl J Med	HCAPLUS
Orwoll, E	2003	18	9	J Bone Miner Res	HCAPLUS
Oxlund, H	1993	53	394	Calcif Tissue Int	HCAPLUS
Rittmaster, R	2000	85	2129	J Clin Endocrinol Me	HCAPLUS
Sato, M	1997	138	4330	Endocrinology	HCAPLUS
Shen, V	1992	50	214	Calcif Tissue Int	HCAPLUS
Shen, V	1998	13	883	J Bone Miner Res	HCAPLUS
Skripitz, R	2000	71	619	Acta Orthop Scand	MEDLINE
Skripitz, R	2001	292	427	Clin Orthop	
Skripitz, R	2000	82-B	138	J Bone Joint Surg Br	
Skripitz, R	2001	83-B	437	J Bone Joint Surg Br	
Takano, Y	1996	11	169	J Bone Miner Res	HCAPLUS
Turner, C	1993	14	595	Bone	MEDLINE
Whitfield, J	1998	63	423	Calcif Tissue Int	HCAPLUS
Wronski, T	1993	132	823	Endocrinology	HCAPLUS
Yamamoto, N	1993	23	333	Bone Miner	HCAPLUS

L18 ANSWER 5 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 2004:117205 HCAPLUS
DN 140:164240
TI Synthesis of selectively labeled peptides using solid-phase peptide synthesis and orthogonal protection schemes
IN Koglin, Norman; Lang, Manja; Beck-Sickinger, Annette G.
PA Universitaet Leipzig, Germany
SO Ger. Offen., 25 pp.
CODEN: GWXXBX
DT Patent
LA German
FAN.CNT 1

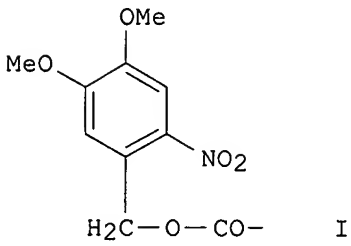
	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	DE 10334499	A1	20040212	DE 2003-10334499	20030723
	WO 2004014941	A1	20040219	WO 2003-DE2534	20030723
	WO 2004014941	C1	20040527		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG,

PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR,
TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

	AU 2003258472	A1	20040225	AU 2003-258472	20030723
	US 2004197824	A1	20041007	US 2003-653779	20030902
PRAI	DE 2002-10233285	A1	20020723		
	US 2002-407179P	P	20020830		
	WO 2003-DE2534	W	20030723		

GI



I

AB A method for the production of specifically N-labeled, especially radiog. labeled, peptides, using orthogonal protection schemes, preferential protective group acid/base cleavage techniques and solid-phase synthetic methods, followed by photolysis of remaining photolabel protecting groups after cleavage from solid-support, is claimed. Labeling can occur on Lys-ε-amines or peptide N-terminal amine groups. Synthetic examples of neuropeptide Y (NPY) and parathyroid hormone labeling using [2,3-3H]-propionic acid, activated as the N-succinimide ester, were given. The photolabile protective group nitroveratryloxycarbonyl [Nvoc] (I) was used to protect non-labeled amines prior to support cleavage, and removed photolytically. Thus, NPY was synthesized using solid-phase techniques, and prior to cleavage from the support, the N-terminal amine was protected using Nvoc-Cl. After support cleavage, the protected peptide was reacted with N-succinimidyl-[2,3-3H]-propionate to give the labeled, protected peptide. Photolysis of the Nvoc group, followed by chromatog. purification, gave the desired labeled peptide, which gave good results in competitive binding studies with Y-receptors Y1, Y2, and Y5 using 'cold' peptide.

IT **83139-29-1**
RL: RCT (Reactant); RACT (Reactant or reagent)
(preparation of selectively labeled peptides using solid-phase peptide synthesis and orthogonal protection schemes)

L18 ANSWER 6 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 2003:931404 HCAPLUS
DN 139:375612
TI Synthesis and therapeutic uses of PTH derivatives resistant to skin proteases
IN Krishna, G. Peri; High, Kim; Bergeron, Annie; Moffett, Serge; Abribat, Thierry
PA Theratechnologies Inc., Can.
SO PCT Int. Appl., 32 pp.
CODEN: PIXXD2
DT Patent

LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2003097690	A2	20031127	WO 2003-CA739	20030516
	WO 2003097690	A3	20040318		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2003233286	A1	20031202	AU 2003-233286	20030516
	US 2004023882	A1	20040205	US 2003-440473	20030516
PRAI	US 2002-378072P	P	20020516		
	WO 2003-CA739	W	20030516		

OS MARPAT 139:375612
AB Protease-resistant analogs of biol. active derivs. of human PTH are described. These analogs are intended for use in therapeutic prepns. for the treatment of various medical conditions in which bone loss is encountered or is susceptible of being encountered. The analogs specified are hPTH(1-34) and hPTH(1-31). More particularly, protease-resistant analogs of PTH adapted for transdermal administration are described.
IT **83139-29-1P**
RL: PAC (Pharmacological activity); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(synthesis and therapeutic uses of PTH derivs. resistant to skin proteases)

L18 ANSWER 7 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 2003:644521 HCAPLUS
DN 139:391567
TI Relaxant effects of parathyroid hormone and parathyroid hormone-related peptides on oviduct motility in birds and mammals: possible role of nitric oxide
AU Francis, M.; Arkle, M.; Martin, L.; Butler, T. M.; Cruz, M. C.; Opare-Aryee, G.; Dacke, C. G.; Brown, J. F.
CS Division of Pharmacology, University of Portsmouth, Portsmouth, PO1 2DT, UK
SO General and Comparative Endocrinology (2003), 133(2), 243-251
CODEN: GCENA5; ISSN: 0016-6480
PB Elsevier Science
DT Journal
LA English
AB Parathyroid hormone (PTH) and PTH-related peptides (PTHrP) have previously been shown to modulate the contractile state of numerous types of smooth muscle. The effects of N-terminal PTH and PTHrP on spontaneous in vitro contractility of oviducal smooth muscle using tissues from egg-laying Japanese quail (10-15 h post ovulation), 4 and 9 days pregnant mouse uterus were investigated. Myometrial tissues from both species contracted vigorously for several hours, when incubated in organ baths in De Jalon's solution gassed with 5%CO2/95%O2. Contractions were enhanced in high (1.2-2.5 mM) compared with low (0.1-0.5 mM) calcium (Ca) containing media. Bovine PTH(1-34) (bPTH(1-34)), human PTH(1-34 amide) (hPTHrP(1-34) amide), and hPTHrP(1-40) caused similar concentration-related inhibition of contractions

in media containing 1.2 mM Ca over a range of 10-9 to 10-7 M, whereas C-terminal hPTHrP(107-139) was devoid of such activity. Responses to bPTH(1-34) in 4 and 9-day pregnant mouse tissues were similar but hPTHrP(1-40) showed substantial loss of activity in 9-day, compared with 4-day pregnant mouse tissues. Repeated exposure of mouse uterine tissue to the peptides resulted in desensitization of responses. The EC50 responses of mouse tissues were inhibited by the PTH/PTHrP receptor antagonist, hPTHrP(7-34) amide. Responses to bPTH(1-34) were also inhibited by both non-selective and selective neuronal nitric oxide synthase (NOS) inhibitors Nω-nitro-L-arginine Me ester (0.01-1 mM) and 7-nitroindazole (0.01-10 μM), resp. Both NOS inhibitors were more effective in inhibiting bPTH(1-34)-induced relaxation in the absence of L-arginine compared with in the presence of 1 mM L-arginine (a NOS substrate) in the incubation media. It is concluded that relaxant responses to N-terminal PTH and PTHrP peptides are well conserved in oviducal and uterine tissues from avian and mammalian species. The results also suggest that NO may be responsible for mediating relaxant activities of these peptides in pregnant mouse uterine tissue.

IT **83139-29-1**, Human PTH(1-34 amide)
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(nitric oxide role in parathyroid hormone and parathyroid hormone-related peptides relaxant effects on oviduct motility in birds and mammals)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Abdul, B	1998	5A	161	Advances in Organ Bi	
Alderton, W	2001	357	593	Biochem J	HCAPLUS
Ardawi, M	1997	137	402	Eur J Endocrinol	HCAPLUS
Bourdeau, A	1990	258	E549	Am J Physiol	HCAPLUS
Bukoski, R	1995	15	536	Semin Nephrol	HCAPLUS
Care, A	1999	84	665	Exp Physiol	HCAPLUS
Charbon, G	1968	3	275	Eur J Pharmacol	HCAPLUS
Chiu, K	1983	74	99	Biochem Physiol	MEDLINE
Collip, J	1925	66	485	J Biol Chem	
Dacke, C	1979			Calcium Regulation i	
Dacke, C	2000		473	Sturkie's Avian Phys	
Ferguson, J	1996	3	338A	J Soc Gynecol Invest	
Ferguson, J	1992	89	8384	Proc Natl Acad Sci U	HCAPLUS
Francis, M	2002	3	P211	Endocrine Abstr	
Jiang, B	1998	31	S142	J Cardiovasc Pharmac	
Kalinowski, L	2001	170	433	J Endocrinol	HCAPLUS
Karperion, M	1996	40	599	Int J Develop Biol	
Kaya, T	1998	37	403	Pharmacol Res	HCAPLUS
Kline, L	2000	91	83	Regulat Pept	HCAPLUS
Kovacs, C	1997	18	832	Endocr Rev	HCAPLUS
Massfelder, T	1996	50	1591	Kidney Int	HCAPLUS
Mok, L	1987	2	329	J Bone Min Res	HCAPLUS
Mok, L	1989	4	433	J Bone Min Res	HCAPLUS
Mosely, J	1996		363	Principles of Bone B	
Nickols, G	1987	24	120	Blood Vessels	HCAPLUS
Nyby, M	1995	136	2497	Endocrinology	MEDLINE
Okano, K	1994	135	1093	Endocrinology	HCAPLUS
Okawa, T	1998	179	721	Am J Obs Gynecol	HCAPLUS
O'Reilly, D	1986	111	501	J Endocrinol	HCAPLUS
Pang, P	1980	77	675	Proc Natl Acad Sci U	HCAPLUS
Pang, P	1986	7	340	Trends Pharmacol Sci	HCAPLUS
Paspaliaris, V	1995	53	259	J Steroid Biochem Mo	HCAPLUS
Quan-Sheng, D	1989	257	E118	Am J Physiol	MEDLINE

Ringer, S	1887	8	288	J Physiol	
Schermer, D	1994	9	1041	J Bone Mineral Res	HCAPLUS
Schleiffer, R	1995	27	415	Hormone Metab Res	HCAPLUS
Shew, R	1991	6	955	J Bone Mineral Res	HCAPLUS
Simeoni, U	1994	86	245	Clin Sci	HCAPLUS
Singh, R	1986	61	20	Gen Comp Endocrinol	HCAPLUS
Sladek, S	1997	272	R441	Am J Physiol	HCAPLUS
Smith, M	1997	20		Bone	
Sutliff, R	1999	140	2077	Endocrinology	HCAPLUS
Thiede, M	1991	129	1958	Endocrinology	HCAPLUS
Urena, P	1993	133	617	Endocrinology	HCAPLUS
van de Velde, J	1984	115	1901	Endocrinology	HCAPLUS
Williams, E	1995	16	205S	Bone	
Williams, E	1994	102	209	J Reprod Fertil	HCAPLUS
Yamamoto, M	1992	89	5326	Proc Natl Acad Sci U	HCAPLUS

L18 ANSWER 8 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 2002:692533 HCAPLUS
DN 138:331836
TI Highly potent analogs of human parathyroid hormone and human parathyroid hormone-related protein
AU Dong, Jesse Z.; Shen, Yeelana; Culler, Michael; Taylor, John E.; Woon, Chee-Wai; Legrand, Jean-Jacques; Morgan, Barry; Chorev, Michael; Rosenblatt, Michael; Nakamoto, Chizu; Moreau, Jacques-Pierre
CS Biomeasure Incorporated/Beaufour-IPSEN, Milford, MA, 01757, USA
SO Peptides: The Wave of the Future, Proceedings of the Second International and the Seventeenth American Peptide Symposium, San Diego, CA, United States, June 9-14, 2001 (2001), 668-669. Editor(s): Lebl, Michal; Houghten, Richard A. Publisher: American Peptide Society, San Diego, Calif.
CODEN: 69DBAL; ISBN: 0-9715560-0-8
DT Conference
LA English
AB Novel human parathyroid hormone (hPTH)/PTH-related protein analogs containing aminoisobutyric acid and 1-aminocyclohexane-1-carboxylic acid, were designed and examined for their effect on cAMP in human osteosarcoma cells. These novel hPTH and hPTHrP analogs were evaluated in rats for their ability of elevating blood calcium concentration Of these compds., one compound showed much lower tendency to mobilize calcium than hPTH(1-34), a property that would contribute to a wider therapeutic index. This compound is about two-fold more efficacious than hPTH (1-34) in restoring femoral bone mineral d. in the OVX, osteopenic rats. At the doses of 1 and 10 μg/kg/day, this compound significantly enhanced bone formation at trabecular sites without affecting the cortical porosity.
IT **83139-29-1D**, Human parathyroid hormone 1-34 amide, analogs
RL: PAC (Pharmacological activity); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(parathormone and parathormone-related protein analogs bone anabolic and calcium mobilizing activity in relation to structure)
RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Culler, M	2001			Am Soc Bone Mineral	
Legrand, J	2001			Am Soc Bone Mineral	
Pellegrini, M	1998	273	10420	J Biol Chem	HCAPLUS
Toniolo, C	1993	11	10	Janssen Chim Acta	HCAPLUS

L18 ANSWER 9 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN

AN 2001:380448 HCAPLUS
DN 135:10001
TI Iontophoretic transdermal delivery of peptides
IN Singh, Parminder Bobby; Liu, Puchun
PA Novartis A.-G., Switz.; Novartis-Erfindungen Verwaltungsgesellschaft m.b.H.
SO PCT Int. Appl., 13 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001036039	A2	20010525	WO 2000-EP11318	20001115
WO 2001036039	A3	20020307		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRAI US 1999-166150P P 19991117

AB A method is disclosed for iontophoretically delivering a drug to a mammal, wherein the drug delivery profile may be adjusted by adjusting the concentration of electrolyte in the reservoir solution of the iontophoretic system. An iontophoretic patch containing 3.0 mg/mL PTA-893 in a Sontara matrix was administered to mini-pigs. The iontophoretic delivery provided a drug delivery profile substantially similar to s.c. injection.
IT **83139-29-1**
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(iontophoretic transdermal delivery of peptides)

L18 ANSWER 10 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 2001:196738 HCAPLUS
DN 134:305606
TI Stimulation of protein kinase C activity in cells expressing human parathyroid hormone receptors by C- and N-terminally truncated fragments of parathyroid hormone 1-34
AU Whitfield, J. F.; Isaacs, R. J.; Chakravarthy, B.; Maclean, S.; Morley, P.; Willick, G.; Divieti, P.; Bringhurst, F. R.
CS Institute for Biological Sciences, National Research Council of Canada, Ottawa, ON, Can.
SO Journal of Bone and Mineral Research (2001), 16(3), 441-447
CODEN: JBMREJ; ISSN: 0884-0431
PB American Society for Bone and Mineral Research
DT Journal
LA English
AB The parathyroid hormone (PTH) fragment PTH(1-34) stimulates adenylyl cyclase, phospholipase C (PLC), and protein kinase C's (PKCs) in cells that express human, opossum, or rodent type 1 PTH/PTH-related protein (PTHrP) receptors (PTHR1s). Certain carboxyl (C)-terminally truncated fragments of PTH(1-34), such as human PTH(1-31) [hPTH-(1-31)NH2], stimulate adenylyl cyclase but not PKCs in rat osteoblasts or PLC and PKCs in mouse kidney cells. The hPTH(1-31)NH2 peptide does fully stimulate PLC in HKRK B7 porcine renal epithelial cells that express 950,000 transfected hPTHR1s per cell. Amino (N)-terminally truncated fragments, such as

bovine PTH(3-34) [bPTH(3-34)], hPTH(3-34)NH2, and hPTH(13-34), stimulate PKCs in Chinese hamster ovary (CHO) cells expressing transfected rat receptors, opossum kidney cells, and rat osteoblasts, but an intact N terminus is needed to stimulate PLC via human PTHR1s in HKRK B7 cells. The authors now report that the N-terminally truncated analogs bPTH(3-34)NH2 and hPTH(13-34)OH do activate PKC via human PTHR1s in HKRK B7 cells, although less effectively than hPTH(1-34)NH2 and hPTH(1-31)NH2. Moreover, in a homologous human cell system (normal foreskin fibroblasts), these N-terminally truncated fragments stimulate PKC activity as strongly as hPTH(1-34)NH2 and hPTH(1-31)NH2. Thus, it appears that unlike their opossum and rodent equivalent, hPTHR1s can stimulate both PLC and PKCs when activated by C-terminally truncated fragments of PTH(1-34). Furthermore, hPTHR1s, like the PTHR1s in rat osteoblasts, opossum kidney cells, and rat PTHR1-transfected CHO cells also can stimulate PKC activity by a mechanism that is independent of PLC. The efficiency with which the N-terminally truncated PTH peptides stimulate PKC activity depends on the cellular context in which the PTHR1s are expressed.

IT **83139-29-1**, HPTH(1-34)NH2
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(parathormone fragment stimulation of protein kinase C activity in cells expressing human parathyroid hormone receptors)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Azzaqrani, A	1995	270	20004	J Biol Chem	
Azzaqrani, A	1996	271	14931	J Biol Chem	
Barbier, J	1997	40	1373	J Med Chem	HCAPLUS
Bringhurst, F	2000	15	S319	J Bone Miner Res	
Chakravarthy, B	1991	196	144	Anal Biochem	HCAPLUS
Chakravarthy, B	1994	304	809	Biochem J	HCAPLUS
Cole, J	2000	15	S318	J Bone Miner Res	
Derrickson, B	1997	272	F781	Am J Physiol	HCAPLUS
Donahue, H	1988	263	13522	J Biol Chem	HCAPLUS
el Hessni, A	1993	92	183	Mol Cell Endocrinol	HCAPLUS
Fraher, L	1999	84	2739	J Clin Endocrinol Me	HCAPLUS
Friedman, P	1999	140	301	Endocrinology	HCAPLUS
Fujimori, A	1992	130	29	Endocrinology	HCAPLUS
Hanafin, N	1995	105	133	J Invest Dermatol	HCAPLUS
Jouishomme, H	1992	130	53	Endocrinology	HCAPLUS
Jouishomme, H	1994	9	943	J Bone Miner Res	HCAPLUS
LiChong, K	1998	23	S356	Bone	
Mohan, S	2000	27	471	Bone	HCAPLUS
Pun, K	1989	259	785	Biochem J	HCAPLUS
Pun, K	1995	4	19	Biol Signals	HCAPLUS
Ribeiro, C	1994	266	F497	Am J Physiol	MEDLINE
Rihani-Bisharat, S	1998	139	974	Endocrinology	HCAPLUS
Rixon, R	1994	9	1179	J Bone Miner Res	HCAPLUS
Siegfried, G	1995	136	1267	Endocrinology	HCAPLUS
Silve, C	1985	60	1144	J Clin Endocrinol Me	HCAPLUS
Singh, A	1999	140	131	Endocrinology	HCAPLUS
Takasu, H	1999	38	13453	Biochemistry	HCAPLUS
Takasu, H	1998	139	4293	Endocrinology	HCAPLUS
Takasu, H	1999	14	11	J Bone Miner Res	HCAPLUS
Usdin, T	1999	2	941	Nat Neurosci	HCAPLUS
Whitfield, J	1999	11	159	Cell Signal	HCAPLUS
Whitfield, J	1999	9	1293	Exp Opin Invest Drug	
Whitfield, J	1997	12	1246	J Bone Miner Res	HCAPLUS
Whitfield, J	2000	15	964	J Bone Miner Res	HCAPLUS

Whitfield, J	1992	150	299	J Cell Physiol	HCAPLUS
Whitfield, J	1999			The Parathyroid Horm	
Wu, T	1987	65	105	J Clin Endocrinol Me	HCAPLUS
Wu, Y	2000	15	879	J Bone Miner Res	HCAPLUS

L18 ANSWER 11 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 2000:284292 HCAPLUS
DN 133:38509
TI The Stimulation of Vertebral and Tibial Bone Growth by the Parathyroid Hormone Fragments, hPTH-(1-31)NH2, [Leu27]cyclo(Glu22-Lys26)hPTH-(1-31)NH2, and hPTH-(1-30)NH2
AU Whitfield, J. F.; Morley, P.; Fraher, L.; Hodsman, A. B.; Holdsworth, D. W.; Watson, P. H.; Willick, G. E.; Barbier, J.-R.; Gulam, M.; Isaacs, R. J.; MacLean, S.; Ross, V.
CS Institute for Biological Sciences, National Research Council of Canada, Ottawa, ON, K1A OR6, Can.
SO Calcified Tissue International (2000), 66(4), 307-312
CODEN: CTINDZ; ISSN: 0171-967X
PB Springer-Verlag New York Inc.
DT Journal
LA English
AB The native human parathyroid hormone, hPTH-(1-84), and certain carboxyl truncated analogs such as hPTH-(1-34) and even smaller fragments such as hPTH-(1-31)NH2, [Leu27]cyclo(Glu22-Lys26)hPTH-(1-31)NH2, and hPTH-(1-30)NH2 stimulate femoral trabecular and cortical bone growth in ovariectomized (OVX) rats. When injected once daily for 6 wk starting 2 wk after OVX in doses of 1 or 2 nmol/100 g of body weight, hPTH-(1-31)NH2, [Leu27]cyclo(Glu22-Lys26)hPTH-(1-31)NH2, and hPTH-(1-34)NH2 prevented the loss of trabecular volume in the L5 vertebrae induced by OVX. In fact, by the end of the sixth week of injections (i.e., the eighth week after OVX) the fragments had increased the volume and trabecular thickness significantly above the values in vehicle-injected sham-operated rats. HPTH-(1-30)NH2 can stimulate vertebral bone growth as much as the larger fragments, but 10-25 times more of it was needed to do so. The same daily doses of hPTH-(1-31)NH2, [Leu27]cyclo(Glu22-Lys26)hPTH-(1-31)NH2, and hPTH-(1-34)NH2 also raised the trabecular volume and thickness in the L5 vertebrae of rats well above the values in vehicle-treated animals when the injections were started 9 wk after OVX. This restoration of trabecular bone in the L5 vertebrae in estrogen-deprived animals was accompanied by a significant increase in the bone mineral d. (BMD) of the L1-L4 vertebrae and tibias. However, there was no significant drop in the pelvic BMD in the estrogen-deprived animals and the effects of hPTH-(1-31)NH2, [Leu27]cyclo(Glu22-Lys26) hPTH-(1-31)NH2, and hPTH-(1-34)NH2 on the pelvic BMD were equivocal.
IT **83139-29-1**, HPTH-(1-34)NH2
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(vertebral, femoral and tibial bone growth stimulation by parathyroid hormone fragments, hPTH-(1-31)NH2, [Leu27]cyclo(Glu22-Lys26)hPTH-(1-31)NH2, hPTH-(1-34)NH2 and hPTH-(1-30)NH2)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Andreassen, T	1999	14	960	J Bone Miner Res	HCAPLUS
Cosman, F	1998	13	1051	J Bone Miner Res	HCAPLUS
Dempster, D	1993	14	690	Endocrine Rev	HCAPLUS
Fraher, L	1999	84	2739	J Clin Endocrinol Me	HCAPLUS
Hodsman, A	1998		83	Anabolic treatments	HCAPLUS

Hodsman, A	1998	23	S631	Bone	
Lindsay, R	1997	350	550	Lancet	HCAPLUS
Mohan, S	1998	23	S449	Bone	
Mosekilde, L	1998		31	Anabolic treatments	HCAPLUS
Whitfield, J	1995	56	227	Calcif Tissue Int	HCAPLUS
Whitfield, J	1996	58	81	Calcif Tissue Int	HCAPLUS
Whitfield, J	1999	65	143	Calcif Tissue Int	HCAPLUS
Whitfield, J	1998			The parathyroid horm	
Wronski, T	1998		59	Anabolic treatments	HCAPLUS
Wronski, T	1998	23	S17	Bone	

L18 ANSWER 12 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 1999:793816 HCAPLUS
DN 132:132462
TI Ca2+ and Extracellular Acidification Rate Responses to Parathyroid Hormone Fragments in Rat ROS 17/2 and Human SaOS-2 Cells
AU Belinsky, Glenn S.; Morley, Paul; Whitfield, James F.; Tashjian, Armen H., Jr.
CS Department of Cancer Cell Biology, Harvard School of Public Health-Harvard Medical School, Boston, MA, 02115, USA
SO Biochemical and Biophysical Research Communications (1999), 266(2), 448-453
CODEN: BBRCA9; ISSN: 0006-291X
PB Academic Press
DT Journal
LA English
AB To examine the importance of the N- or C-termini of PTH(1-34) the effects of truncated fragments of PTH on human receptors in osteoblast-like SaOS-2 cells and rat receptors in rats ROS 17/2 cells were examined Fura-2-loaded cells were used to monitor cytosolic free Ca2+ concentration ([Ca2+]i), and the Cytosensor microphysiometer was used to monitor extracellular acidification rate (ECAR). C-terminally truncated fragments (1-31) and (1-28) of hPTH(1-34)NH2 stimulated an increase in [Ca2+]i and ECAR in both cell lines. HPTH(3-34)NH2 and other N-terminally truncated fragments did not stimulate [Ca2+]i or ECAR in either cell type. The signal transduction pathway of PTH-induced ECAR in ROS 17/2 cells was investigated to compare with previous results in SaOS-2 cells. Potentiation by IBMX, attenuation by 8Br-cAMP and lack of effect of the PKC inhibitor chelerythrine chloride support a cAMP/PKA-mediated signal transduction pathway in ROS 17/2, while the protein kinase C pathway was predominant in SaOS-2 cells. The authors conclude that the intact N-terminus of PTH is essential in PTH signaling mediated via either the cAMP/PKA or inositol lipid/Ca2+/PKC pathways in osteoblast-like cells. (c) 1999 Academic Press.
IT **83139-29-1**, Human parathyroid hormone 1-34 amide
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(N-terminus of parathyroid hormone is essential for several PTH signaling pathways in rat ROS 17/2 and human SaOS-2 cells)
RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Abou-Samra, A	1989	124	1107	Endocrinology	HCAPLUS
Armamento-Villareal, R	1997	12	384	J Bone Miner Res	HCAPLUS
Barrett, M	1997	272	26346	J Biol Chem	HCAPLUS
Belinsky, G	1999			J Bone Miner Res in	
Bidwell, J	1991	129	2993	Endocrinology	HCAPLUS
Boland, C	1986	118	980	Endocrinology	HCAPLUS
Bushinsky, D	1996	271	F216	Am J Physiol	HCAPLUS

Bushinsky, D	1994	20	40	Miner Electrolyte Me	MEDLINE
Chen, L	1999	11	499	Cell Signal	HCAPLUS
Coldwell, M	1999	127	1135	Br J Pharmacol	HCAPLUS
Donahue, H	1988	263	13522	J Biol Chem	HCAPLUS
Dunlay, R	1990	258	F223	Am J Physiol	HCAPLUS
Fujimori, A	1991	128	3032	Endocrinology	HCAPLUS
Grynkiewicz, G	1985	260	3440	J Biol Chem	HCAPLUS
Hodsman, A	1990	9	137	Bone Miner	MEDLINE
Jouishomme, H	1992	130	53	Endocrinology	HCAPLUS
Jouishomme, H	1994	9	943	J Bone Miner Res	HCAPLUS
Juppner, H	1991	254	1024	Science	HCAPLUS
Lowik, C	1985	6	311	Cell Calcium	MEDLINE
McConnell, H	1992	257	1906	Science	HCAPLUS
Owicki, J	1990	87	4007	Proc Natl Acad Sci	HCAPLUS
Parce, J	1989	246	243	Science	HCAPLUS
Potts, J	1995		920	Endocrinology	
Reeve, J	1976	1	1035	Lancet	MEDLINE
Rixon, R	1994	9	1179	J Bone Miner Res	HCAPLUS
Schofl, C	1991	274	15	Biochem J	
Whitfield, J	1999	65	143	Calcif Tissue Int	HCAPLUS
Whitfield, J	1997	12	1246	J Bone Miner Res	HCAPLUS
Wiltink, A	1993	14	591	Cell Calcium	HCAPLUS

L18 ANSWER 13 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 1999:479995 HCAPLUS
DN 131:252837
TI Stimulation of Femoral Trabecular Bone Growth in Ovariectomized Rats by Human Parathyroid Hormone (hPTH)-(1-30)NH2
AU Whitfield, J. F.; Morley, P.; Willick, G. E.; MacLean, S.; Ross, V.; Barbier, J.-R.; Isaacs, R. J.
CS Building M-54, Institute for Biological Sciences, National Research Council of Canada, Ottawa, ON, K1A 0R6, Can.
SO Calcified Tissue International (1999), 65(2), 143-147
CODEN: CTINDZ; ISSN: 0171-967X
PB Springer-Verlag New York Inc.
DT Journal
LA English
AB It has been proposed that intermittent bursts of adenylyl cyclase and the surges of cAMP they produce can trigger PTH's bone anabolic action without the activation of phospholipase-C (PLC). This was based on the osteogenic action in ovariectomized (OVX) rats of hPTH-(1-31)NH2, which can stimulate adenylyl cyclase but not PLC in ROS 17/2 rat osteosarcoma cells, and the osteogenic impotence of fragments such as 1-desamino-hPTH-(1-34) and hPTH-(8-84) which strongly stimulate PLC but not adenylyl cyclase. But this seems to have been disproven by the inability of hPTH-(1-30)NH2 to stimulate bone growth despite its having hPTH-(1-31)NH2's ability to strongly stimulate adenylyl cyclase but not PLC in cells with rat type 1 PTH/PTHrP receptors. Because of the importance of hPTH-(1-30)NH2's apparent osteogenic impotence for knowing how PTH triggers bone growth, we have reinvestigated the fragment's ability to stimulate trabecular bone growth in the femurs of young OVX rats and have found it to be strongly osteogenic at doses 2-10 times higher than the highest dose used previously. Thus, 6 wk of once-daily s.c. injections of 10-50 nmol of hPTH-(1-30)NH2/100 g into young rats starting 2 wk after OVX significantly increased the femoral trabecular volume and mean thickness of individual trabeculae above those in sham-operated control rats. In OVX rats treated with 50 nmol of hPTH-(1-30)NH2/100 g, the trabecular volume was 2.6 times higher and the mean trabecular thickness nearly 4 times higher than in the sham-operated control rats. This very large increase in the mean trabecular thickness was as much as the increase induced by 2 nmol/100 g

of hPTH-(1-31)NH2, [Leu27]cyclo(Glu22-Lys26)-hPTH-(1-31)NH2, hPTH-(1-34)NH2 and [Leu27]cyclo(Glu22-Lys26)-hPTH-(1-34)NH2. These results have removed a major objection to the proposal that PTH's osteogenic action in rats can be triggered solely by intermittent surges of cAMP and the bursts of cAMP-dependent protein kinase activity they cause.

IT **83139-29-1**, HPTH-(1-34)NH2
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(human parathyroid hormone fragment stimulation of femoral trabecular bone growth in ovariectomized rats and mechanism therefor)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Barbier, J	1997	40	1373	J Med Chem	HCAPLUS
Dempster, D	1995	16	157	Bone	MEDLINE
Friedman, P	1999	140	301	Endocrinology	HCAPLUS
Jouishomme, H	1994	9	943	J Bone Miner Res	HCAPLUS
Miller, B	1997	12	S320	J Bone Miner Res	
Neugebauer, W	1995	34	8835	Biochemistry	HCAPLUS
Rixon, R	1994	9	1179	J Bone Miner Res	HCAPLUS
Strein, K	1994	12	131	Bone disease and ost	
Takasu, H	1998	139	4293	Endocrinology	HCAPLUS
Takasu, H	1997	12	S444	J Bone Miner Res	
Whitfield, J	1998			Anabolic treatments	
Whitfield, J	1996	58	81	Calcif Tissue Int	HCAPLUS
Whitfield, J	1997	60	302	Calcif Tissue Int	HCAPLUS
Whitfield, J	1999	11	159	Cell Signal	HCAPLUS
Whitfield, J	1997	12	1246	J Bone Miner Res	HCAPLUS
Whitfield, J	1998			The parathyroid horm	

L18 ANSWER 14 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 1999:396580 HCAPLUS
DN 131:194463
TI Secondary and tertiary fold of the human parathyroid hormone 1-34 in different environments as a pathway to bioactive conformation
AU Pellegrini, Maria; Maretto, Stefano; Royo, Miriam; Rosenblatt, Michael; Chorev, Michael; Mierke, Dale F.
CS Chemistry Department, Clark University, Worcester, MA, 01610, USA
SO Peptides: Frontiers of Peptide Science, Proceedings of the American Peptide Symposium, 15th, Nashville, June 14-19, 1997 (1999), Meeting Date 1997, 402-403. Editor(s): Tam, James P.; Kaumaya, Pravin T. P. Publisher: Kluwer, Dordrecht, Neth.
CODEN: 67UCAR
DT Conference
LA English
AB The present study details the conformation of hPTH(1-34)-NH2, investigated by CD and NMR spectroscopy in different environments and under varying exptl. conditions. The tendency of the peptide to fold into α-helix in two segments is therefore clear from the three studies. The DPC micelles seem to favor greatly this secondary structure, indicating that the interaction of the peptide at the membrane interface where the receptor is embedded may play a major role in inducing the bioactive conformation.
IT **83139-29-1**, Human parathyroid hormone 1-34 amide
RL: PRP (Properties)
(secondary and tertiary fold of human parathyroid hormone 1-34 in different environments as pathway to bioactive conformation)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Marx, U	1995	270	1519	J Biol Chem	
Mierke, D	1994	44	325	Int J Pept Protein r	HCAPLUS
Moroder, L	1993	32	13551	J Biochemistry	HCAPLUS

L18 ANSWER 15 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 1999:276855 HCAPLUS
DN 131:83086
TI Stimulation of membrane-associated protein kinase-C activity in spleen lymphocytes by hPTH-(1-31) NH2, its lactam derivative, [Leu27]-cyclo(Glu22-Lys26)-hPTH-(1-31) NH2, and hPTH-(1-30) NH2
AU Whitfield, James F.; Isaacs, Rick; MacLean, Sue; Morley, Paul; Barbier, Jean-Rene; Willick, Gordon E.
CS Institute for Biological Sciences, National Research Council of Canada, Ottawa, ON, K1A 0R6, Can.
SO Cellular Signalling (1999), 11(3), 159-164
CODEN: CESIEY; ISSN: 0898-6568
PB Elsevier Science Inc.
DT Journal
LA English
AB Human parathyroid hormone, hPTH-(1-34), stimulates adenylyl cyclase and phosphatidylinositol-bisphosphate-specific phospholipase-C (PIP2-PLC), as indicated by increased membrane-associated protein kinase C (PKC) activity in ROS 17/2 rat osteosarcoma cells. The C-terminally truncated hPTH-(1-31)NH2 stimulates adenylyl cyclase as strongly as hPTH-(1-34) in these cells, but it does not stimulate PKC activity. Even [Leu27]-cyclo(Glu22-Lys26)-hPTH-(1-31)NH2, a 6-fold stronger adenylyl cyclase stimulator than hPTH-(1-34), cannot stimulate PKC activity in ROS cells. Therefore PTH required its 32-34 region to stimulate PIP2-PLC/PKCs in this osteosarcoma line. In contrast, hPTH-(1-31)NH2 [Leu27]-cyclo(Glu22-Lys26)-hPTH-(1-31)NH2 and even hPTH-(1-30)NH2 can stimulate PKC activity in freshly isolated rat spleen lymphocytes as strongly as hPTH-(1-34)NH2. The difference in the ability of membrane-associated PKC activity in spleen lymphocytes, but not in ROS cells, to be stimulated by C-terminally truncated PTH fragments might be due to different receptor densities or to the lymphocyte's atypical PTH/PTHrP receptor.
IT **83139-29-1**, HPTH-(1-34)-NH2
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(parathormone fragment differential stimulation of membrane-associated protein kinase C in spleen lymphocytes and ROS cells)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Atkinson, M	1987	2	303	J Bone Miner Res	HCAPLUS
Barbier, J	1997	40	1373	J Med Chem	HCAPLUS
Bergwitz, C	1996	271	26469	J Biol Chem	HCAPLUS
Bos, M	1996	58	95	Calcif Tissue Int	HCAPLUS
Chakravarthy, B	1991	196	144	Anal Biochem	HCAPLUS
Chakravarthy, B	1990	171	1105	Biochem Biophys Res	HCAPLUS
Chakravarthy, B	1994	304	809	Biochem J	HCAPLUS
Friedman, P	1997	12	S317	J Bone Miner Res	
Guo, J	1995	136	3884	Endocrinology	HCAPLUS
Huang, Z	1996	271	33382	J Biol Chem	HCAPLUS
Iida-Klein, A	1997	272	6882	J Biol Chem	HCAPLUS
Jouishomme, H	1992	130	53	Endocrinology	HCAPLUS

Jouishomme, H	1994	9	943	J Bone Miner Res	HCAPLUS
Neugebauer, W	1995	34	8835	Biochemistry	HCAPLUS
Offermanns, S	1996	10	566	Mol Endocrinol	HCAPLUS
Orloff, J	1995	136	3016	Endocrinology	HCAPLUS
Rixon, R	1994	9	1179	J Bone Miner Res	HCAPLUS
Schluter, K	1995	310	439	Biochem J	
Takasu, H	1997	12	S444	J Bone Miner Res	
Whitfield, J	1998		109	Anabolic Treatments	HCAPLUS
Whitfield, J	1996	58	81	Calcif Tissue Int	HCAPLUS
Whitfield, J	1997	12	1246	J Bone Miner Res	HCAPLUS
Whitfield, J	1971	78	355	J Cell Physiol	HCAPLUS
Whitfield, J	1992	150	299	J Cell Physiol	HCAPLUS
Whitfield, J	1994	158	518	J Cell Physiol	HCAPLUS

L18 ANSWER 16 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 1998:282392 HCAPLUS
DN 128:321948
TI Continuous low-dose administration of parathyroid hormone or its agonist
IN Chorev, Michael; Rosenblatt, Michael
PA Beth Israel Deaconess Medical Center, USA
SO U.S., 6 pp., Cont.-in-part of U.S. Ser. No. 359,293, abandoned.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 5747456	A	19980505	US 1996-724539	19960930
	CA 2205959	AA	19960627	CA 1995-2205959	19951219
	WO 9814478	A1	19980409	WO 1997-US17216	19970925
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	AU 9745016	A1	19980424	AU 1997-45016	19970925
PRAI	US 1994-359293	B2	19941219		
	US 1996-724539	A	19960930		
	WO 1997-US17216	W	19970925		

OS MARPAT 128:321948
AB A method of promoting bone formation in a human patient, which includes the step of administering continuously to the patient parathyroid hormone or its agonist, e.g. 1-34-human PTH-NH2, 1-34-[Nle8,18,Tyr34]-human PTH-NH2, 1-34-bovine PTH-NH2, 1-34-[Nle8,18,Tyr34]-bovine PTH-NH2, 1-34-[Nle8,18,Phe22,Tyr34]-bovine PTH-NH2, 1-34-[Nle8,18,Arg19,Tyr34]-bovine PTH-NH2, 1-34-[Nle8,18,Arg21,Tyr34]-bovine PTH-NH2, 1-34-[Nle8,18,Arg19,21,Tyr34]-bovine PTH-NH2, for a period of at least one month at a dosage between 10 and 400 units/24 h. Also disclosed are novel parathyroid hormone agonists R1R2A-Val-Ser-Glu-Ile-Gln-A7-Nle-His-Asn-Leu-A12-Lys-His-Leu-A16-Ser-Nle-A19-Asn-A21-A22-A23-Leu-Arg-Lys-Lys-Leu-Gln-Asp-Val-His-Asn-A34-W [A1 = Ser, Ala; A7 = Leu, Phe; A12 = Gly, Aib, Ala, D-Ala; A16 = Asn, Ser Ala; A19 = Glu, Arg, Lys, Asp, Ser, Thr, Gln, Asn, Ala; A21 = Val, Met, Arg, Lys, Glu, Asp, Ser, Thr, Gln, Asn, Leu, Ile, Nle, Ala, Phe, p-X-Phe, X = OH, Me, NO2, halo; A22 = Glu, Asp, Phe, p-X-Phe Ser, Thr, Gln, Asn, Leu, Ile, Nle, Val, Ala, Met; A23 = Trp, 1-Nal, 2-Nal; W = OH, C1-12 alkoxy, C7-20 phenylalkoxy, C11-20 naphthylalkoxy, NR3R4; with proviso that when A12 = Gly, A19 = Glu, A21 =

Val, A22 = Gln, then A23 = 1-Nal or 2-Nal; each R1, R2, R3, R4 = independently H, C1-12 alkyl, C7-10 phenylalkyl, CO-E; E = C1-12 alkyl, C2-12 alkenyl, Ph, naphthyl, C7-20 phenylalkyl] or a pharmaceutically acceptable salt thereof.

IT **83139-29-1P**, 1-34-Human PTH-NH2
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation and continuous low-dose administration of parathyroid hormone or its agonist)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Anon	1992			EP 0477885 A2	HCAPLUS
Dempster	1993	14	690	Endocrine Reviews	HCAPLUS
Dempster	1993	14	690	Endocrinse Reviews	HCAPLUS
Hesch	1989	44	176	Calcified Tissue Int	MEDLINE
Liu	1990	5	973	Journal of Bone and	HCAPLUS
Malluche	1982		F197	The American Physiol	HCAPLUS
Morita	1987			US 4656250	HCAPLUS
Podbesek	1983		1000	Endocrinology	HCAPLUS
Reeve	1980		1340	British Medical Jour	MEDLINE
Rosenblatt	1988			US 4771124	HCAPLUS

L18 ANSWER 17 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 1998:268933 HCAPLUS
DN 128:239555
TI New insights into interactions between the human PTH/PTHrP receptor and agonist/antagonist binding
AU Fukyayama, Shoichi; Royo, Miryam; Sugita, Masahiko; Imrich, Amy; Chorev, Michael; Suva, Larry J.; Rosenblatt, Michael; Tashjian, Armen H., Jr.
CS Dep. of Molecular and Cellular Toxicology, Harvard Medical School, Boston, MA, 02115, USA
SO American Journal of Physiology (1998), 274(2, Pt. 1), E297-E303
CODEN: AJPHAP; ISSN: 0002-9513
PB American Physiological Society
DT Journal
LA English
AB We prepared a polyclonal antiserum [Ab-(88-97)] against residues 88-97 of the NH2-terminal tail of the human (h) parathyroid hormone (PTH)/PTH-related protein (PTHrP) receptor. Ab-(88-97) bound specifically to the receptor, as assessed by fluorescence-activated cell sorter anal. of HEK C21 cells, which stably express .apprx.400,000 hPTH/PTHrP receptors per cell. Unlike PTH, Ab-(88-97) binding did not elicit either cAMP or intracellular calcium concentration signaling responses in these cells. Incubation of C21 cells for 90 min at 4° with hPTH-(1-34) plus antiserum reduced the Ab-(88-97) binding to the cells by up to 40-50% of control values in a PTH concentration-dependent fashion with a half-maximal effective concentration of .apprx.5 nM. The decrease in Ab-(88-97) binding caused by hPTH-(1-34) was completely reversed by coincubation with hPTHrP-(7-34). We conclude that residues 88-97 of the hPTH/PTHrPR are involved, either directly or indirectly, in agonist but not antagonist binding to the receptor.

IT **83139-29-1**, HPTH-(1-34)NH2
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(interactions between human PTH/PTHrP receptor and agonist/antagonist binding in relation to receptor structure)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Abou-Samra, A	1992	89	2732	Proc Natl Acad Sci U	HCAPLUS
Behar, V	1996	137	2748	Endocrinology	HCAPLUS
Buggy, J	1996	28	215	Horm Metab Res	HCAPLUS
Chorev, M	1994		139	The Parathyroids:Bas	HCAPLUS
Dallas, J	1996	137	3329	Endocrinology	HCAPLUS
Fukayama, S	1993	264	C464	Am J Physiol, Cell P	
Fukayama, S	1996	271	C121	Am J Physiol, Cell P	
Fukayama, S	1997	9	469	Cell Signalling	HCAPLUS
Fukayama, S	1992	131	1757	Endocrinology	HCAPLUS
Fukayama, S	1994	134	1851	Endocrinology	HCAPLUS
Gardella, T	1996	271	19888	J Biol Chem	HCAPLUS
Juppner, H	1994	134	879	Endocrinology	HCAPLUS
Juppner, H	1991	254	1024	Science	HCAPLUS
Larner, R	1981	78	3403	Proc Natl Acad Sci U	
Lee, C	1994	135	1488	Endocrinology	HCAPLUS
Moseley, J	1987	84	5048	Proc Natl Acad Sci U	HCAPLUS
Nutt, R	1990	127	491	Endocrinology	HCAPLUS
Pines, M	1996	18	381	Bone	HCAPLUS
Pines, M	1994	135	1713	Endocrinology	HCAPLUS
Rosenblatt, M	1989		848	Endocrinology	
Rosenblatt, M	1986	315	1004	N Engl J Med	HCAPLUS
Schipani, E	1993	132	2157	Endocrinology	HCAPLUS
Schneider, H	1993	246	149	Eur J Pharmacol	HCAPLUS
Shigeno, C	1988	263	3864	J Biol Chem	HCAPLUS
Strewler, G	1987	80	1803	J Clin Invest	HCAPLUS
Stringer, B	1995	20	23	Cytometry	MEDLINE
Suva, L	1987	237	893	Science	HCAPLUS
Unson, C	1996	93	310	Proc Natl Acad Sci U	HCAPLUS
Usdin, T	1995	270	15455	J Biol Chem	HCAPLUS
Yamamoto, I	1988	122	1208	Endocrinology	HCAPLUS
Yamamoto, S	1997	138	2066	Endocrinology	HCAPLUS
Zhou, A	1997	94	3644	Proc Natl Acad Sci U	HCAPLUS

L18 ANSWER 18 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 1998:219833 HCAPLUS
DN 128:283090
TI Preparation and continuous low-dose administration of parathyroid hormone or its agonist as bone formation promoters
IN Chorev, Michael; Rosenblatt, Michael
PA Beth Israel Deaconess Medical Center, USA; Chorev, Michael; Rosenblatt, Michael
SO PCT Int. Appl., 27 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9814478	A1	19980409	WO 1997-US17216	19970925

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

	US 5747456	A	19980505	US 1996-724539	19960930
	AU 9745016	A1	19980424	AU 1997-45016	19970925
PRAI	US 1996-724539	A	19960930		
	US 1994-359293	B2	19941219		
	WO 1997-US17216	W	19970925		

AB A method of promoting bone formation in a human patient, which includes the step of administering continuously to the patient parathyroid hormone or its agonist R1R2A-Val-Ser-Glu-Ile-Gln-A7-Nle-His-Asn-Leu-A12-Lys-His-Leu-A16-Ser-Nle-A19-Asn-A21-A22-A23-Leu-Arg-Lys-Lys-Leu-Gln-Asp-Val-His-Asn-A34-W [A1 = Ser, Ala; A7 = Leu, Phe; A12 = Gly, α-aminoisobutyric acid (Aib), Ala, D-Ala; A16 = Asn, Ser, Ala; A19 = Glu, Arg, Lys, Asp, Ser, Thr, Gln, Asn, Ala; A21 = Val, Met, Arg, Lys, Glu, Asp, Ser, Thr, Gln, Asn, Leu, Ile, Nle, Ala, Phe, p-X-Phe; A22 = any residue A21 except Arg or Lys; A23 = Trp, 3-(1-naphthyl)alanine (1-Nal), 3-(2-naphthyl)alanine (2-Nal); A34 = Phe, p-X-Phe; W = OH, C1-12 alkoxy, C7-20 phenylalkoxy, C11-20 naphthylalkoxy, NR3R4; X = OH, Me, NO2, halo; each R1-R4 = independently H, C1-12 alkyl, C7-10 phenylalkyl, CO-E; E = C1-12 alkyl, C2-12 alkenyl, Ph, naphthyl, C7-20 phenylalkyl; with the proviso that when A12 = Gly, A19 = Glu, A21 = Val, and A22 = Gln, then A23 = 1-Nal or 2-Nal], or a pharmaceutically acceptable salt thereof, for a period of at least one month at a dosage between 10 and 400 units/24 h. Also disclosed are novel parathyroid hormone agonists. Thus, [Nle8,18,Arg21,Tyr34]bPTH1-34-NH2, prepared by standard solid-phase methods using tert-butoxycarbonyl (Boc) chemical, showed IC50 = 0.3 nM in a competitive binding assay using SaOS-2 B10 cells, and EC50 = 0.2 nM in an adenylate cyclase activity assay also measured in SaOS-2 B10 cells.

IT **83139-29-1**, HPTH1-34-NH2
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(preparation and continuous low-dose administration of parathyroid hormone or its agonist as bone formation promoters)

RETABLE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)	Referenced File
Beth Israel Hospital As	1996			WO 9619246 A	HCAPLUS
Morita, K	1987			US 4656250 A	HCAPLUS
Procter & Gamble Pharma	1993			WO 9311786 A	HCAPLUS
Sandoz Ag	1994			WO 9402510 A	HCAPLUS

L18 ANSWER 19 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 1997:542143 HCAPLUS
DN 127:215534
TI Diagnostic reagents of diabetic nephropathy without clinical symptoms
IN Ogata, Etsuro; Kurokawa, Kiyoshi; Uchida, Toshiya; Kishida, Akiko
PA Asahi Chemical Industry Co., Ltd., Japan
SO Jpn. Kokai Tokkyo Koho, 11 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	JP 09208497	A2	19970812	JP 1996-12922	19960129
	WO 2004076688	A1	20040910	WO 1993-JP178	19930212
	W: US				
	US 5662604	A	19970902	US 1995-495635	19950919
PRAI	WO 1993-JP178	W	19930212		
	JP 1996-12922	A	19960129		

AB Diabetic nonsymptomatic nephropathy is diagnosed by measureing the ratio of the increase in urine levels of N-acetyl-β-D-glucosaminidase (NAG) before and after i.v. injection of PTH. The average urinary NAG ration before and after PTH injection was decreased in patients with diabetic nonsymptomatic nephropathy as compared with that in normal healthy subjects (2.3 vs. 5.3). Apparently, early diagnosis of diabetic nonsymptomatic nephropathy may be made by the post PTH decrease in the urine NAG ratio.

IT **83139-29-1**, Human PTH (1-34)-NH2
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(post PTH urinary N-acetyl-β-D-glucosaminidase in diagnosis of nonsymptomatic diabetic nephropathy)

L18 ANSWER 20 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 1996:462564 HCAPLUS
DN 125:105862
TI Continuous low-dose administration of parathyroid hormone or its agonist
IN Chorev, Michael; Rosenblatt, Michael
PA Beth Israel Hospital Association, USA
SO PCT Int. Appl., 23 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9619246	A1	19960627	WO 1995-US16554	19951219
	W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TT				
	RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	CA 2205959	AA	19960627	CA 1995-2205959	19951219
	AU 9644728	A1	19960710	AU 1996-44728	19951219
	EP 800405	A1	19971015	EP 1995-943474	19951219
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE				
	JP 10511095	T2	19981027	JP 1995-519949	19951219
PRAI	US 1994-359293	A	19941219		
	WO 1995-US16554	W	19951219		

AB A method of promoting bone formation in a human patient, which includes the step of administering continuously to the patient parathyroid hormone (PTH) or its agonists, including 1-34-human PTH-NH2, 1-34-[Nle8,18,Tyr34]-human PTH-NH2, 1-34-bovine PTH-NH2, 1-34-[Nle8,18,Tyr34]-bovine PTH-NH2, 1-34-[Nle8,18,Phe22,Tyr34]-bovine PTH-NH2, 1-34-[Nle8,18,Arg19,Tyr34]-bovine PTH-NH2, 1-34-[Nle8,18,Arg21,Tyr34]-bovine PTH-NH2, 1-34-[Nle8,18,Arg19,21,Tyr34]-bovine PTH-NH2, for a period of at least one month at a dosage between 10 and 400 units/24 h, is presented. Also disclosed are novel parathyroid hormone agonists as bone resorption-inhibiting agents.

IT **83139-29-1**, 1-34-Human PTH-NH2
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(parathyroid hormone agonist continuous low-dose administration promotion of bone formation in humans)

L18 ANSWER 21 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 1996:422004 HCAPLUS
DN 125:105556
TI PTH/PTHrP receptor is temporally regulated during osteoblast differentiation and is associated with collagen synthesis
AU McCauley, Laurie K.; Koh, amy J.; Beecher, Christopher A.; Cui, Yinqui; Rosol, Thomas J.; Franceschi, Renny T.
CS Dep. of Periodontics/Prevention/Geriatrics, Univ. of Michigan, Ann Arbor, MI, 48109-1078, USA
SO Journal of Cellular Biochemistry (1996), 61(4), 638-647
CODEN: JCEBD5; ISSN: 0730-2312
PB Wiley-Liss
DT Journal
LA English
AB The temporal sequence of PTH/PTHrP receptor mRNA, binding biol. activity, and its dependence on matrix synthesis was determined using MC3T3-E1 preosteoblast-like cells and primary rat calvarial cells in vitro. Osteoblastic cells were induced to differentiate and form mineralized nodules with the addition of ascorbic acid and β-glycerophosphate, and samples were collected from 9-26 days of culture. DNA levels as determined by fluorometric anal. increased 12- and 17-fold during the collection period for both MC3T3-E1 and primary calvarial cells, resp. Steady state mRNA levels for the PTH/PTHrP receptor as determined by northern blot anal., were initially low for both cell types, peaked at day 4 and 5 for MC3T3-E1 and primary calvarial cells resp., ad declined thereafter. Competition binding curves were performed during differentiation using 125I-PTHrP. The nos. of receptors per μg DNA were greatest at days 3 and 5 for MC3T3-E1 and primary calvarial cells resp. The biol. activity of the receptor was evaluated by stimulating the cells with 10 nM PTHrP and determining cAMP levels via a binding protein assay. The PTHrP-stimulated cAMP levels increased 5-fold to peak values at day 5 for MC3T3-E1 cells and 6-fold to peak values at day 4 for the primary calvarial cells. Ascorbic acid was required for maximal development of a PTH-dependent cAMP response since ascorbic acid-treated MC3T3-E1 cells had twice the PTH-stimulated cAMP levels as non-treated cells. When the collagen synthesis inhibitor 3,4-dehydroproline was administered to MC3T3-E1 cultures prior to differentiation, there was a subsequent diminution of the PTH/PTHrP receptor mRNA gene expression and nos. of receptors per cell; however, if administered after the initiation of matrix synthesis there was no reduction in PTH/PTHrP receptor mRNA. These findings indicate that the PTH/PTHrP receptor is associated temporally at the level of mRNA, protein, and biol. activity, with a differentiating, matrix-producing osteoblastic cell in vitro.
IT **83139-29-1**, 1-34-Human PTH amide
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(parathormone receptor temporal regulation during osteoblast differentiation and association with collagen formation)

L18 ANSWER 22 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 1996:259452 HCAPLUS
DN 125:222445
TI Preparation of parathyroid hormone analogs for treatment of osteoporosis
IN Willick, Gordon E.; Whitfield, James F.; Surewicz, Witold; Sung, Wing L.; Neugebauer, Witold
PA Rixon, Raymond H., Can.
SO Can. Pat. Appl., 21 pp.
CODEN: CPXXEB
DT Patent

LA English
FAN.CNT 4

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	CA 2126299	AA	19951221	CA 1994-2126299	19940620
	CA 2126299	C	20001212		
	US 5556940	A	19960917	US 1994-262495	19940620
PRAI	CA 1994-2126299	A	19940620		

AB Certain analogs of human parathyroid hormone (hPTH) are prepared and have been found to be effective for the treatment of osteoporosis, while showing decreased side effects. Analogs showing this effect include all sequences from hPTH-(1-28)-NH2 to hPTH-(1-31)-NH2 and all sequences from [Leu27]-hPTH-(1-28)-NH2 to [Leu27]-hPTH-(1-34)-NH2, and cyclic analogs cyclo(Lys26-Asp30)[Leu27]-hPTH-(1-34)-NH2 (I) and cyclo(Lys27-Asp30)-hPTH-(1-34)-NH2. Analogs in the form of the carboxyl terminal amide are particularly effective. Thus, I was synthesized with a Milligen 9050 Plus continuous-flow peptide synthesizer on TentaGel S-RAM as the solid support using N-Fmoc-protected amino acids, wherein the side chain amino group of Lys was protected as the Boc derivative and the coupling of Lys26 was accomplished outside of the synthesizer. The resulting peptide-resin was swollen in CH2Cl2 and the tert-Bu group of Asp10 and Boc group of Lys26 were removed by treatment with 305 CF3CO2H in CH2Cl2 for 15 min. The resin was thoroughly washed with solvents and then returned to the synthesizer and cyclization was accomplished by 2 cycles of 3 h-treatment with (benzotriazolyl)-N-oxypyrrolidinium phosphonium hexafluorophosphate/diisopropylethylamine/DMF. The ability of hPTH analogs to bind to receptors and activate the adenyl cyclase coupled signaling mechanism was carried out on a differentiation competent osteoblast-like RCS 17/2 rat osteosarcoma cell line (Whitfield et al, J. Cell Physiol., 1992). I at 7 nM in vitro showed the concentration necessary to express a half (50%)-maximal increase in the adenyl cyclase activity.

IT **83139-29-1P**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of parathyroid hormone analogs for treatment of osteoporosis)

L18 ANSWER 23 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 1995:660848 HCAPLUS
DN 123:247290
TI A comparison of the anabolic effects of parathyroid hormone at skeletal sites with moderate and severe osteopenia in aged ovariectomized rats
AU Qi, H.; Li, M.; Wronski, T.J.
CS College of Veterinary Medicine, University of Florida, Gainesville, FL, USA
SO Journal of Bone and Mineral Research (1995), 10(6), 948-55
CODEN: JBMREJ; ISSN: 0884-0431
DT Journal
LA English
AB Previous studies have shown that parathyroid hormone (PTH) stimulates bone formation and completely restores lost cancellous bone at skeletal sites with moderate osteopenia in relatively young ovariectomized (OVX) rats. The current study was designed to determine whether PTH has similar bone anabolic effects in aged OVX rats and to compare the bone restorative response to PTH at skeletal sites with moderate and severe osteopenia. Female Sprague-Dawley rats were subjected to sham surgery or bilateral ovariectomy at 3 mo of age and maintained untreated for the first year after surgery to allow for the development of moderate vertebral osteopenia and severe tibial osteopenia in OVX rats. Groups of baseline control and OVX rats were sacrificed at the end of this pretreatment

period. The remaining OVX rats were then treated for 10 wk with vehicle, antiresorptive agents alone (estrogen, the bisphosphonate risedronate, or calcitonin) or PTH alone. Other groups of OVX rats were treated concurrently with PTH and each of the antiresorptive agents. As expected, the proximal tibia of baseline OVX rats exhibited severe cancellous osteopenia, whereas the first lumbar vertebral body was moderately osteopenic. Treatment of OVX rats with antiresorptive agents alone failed to restore cancellous bone at both skeletal sites, whereas treatment with PTH alone markedly stimulated bone formation and completely restored lost cancellous bone in the lumbar vertebra. PTH also stimulated bone formation in the severely osteopenic proximal tibia of OVX rats but only marginally restored lost cancellous bone, possibly due to an inadequate number of bone spicules to serve as a foundation for new bone formation. Concurrent treatments with PTH and antiresorptive agents did not augment cancellous bone to a greater, or lesser, extent than treatment with PTH alone. The pos. results from the moderately osteopenic lumbar vertebra indicate that cancellous bone of aged OVX rats retains its ability to respond anabolically to PTH. However, the neg. results from the severely osteopenic proximal tibia of aged OVX rats may provide insight into the failure of the skeletons of some osteoporotic patients to respond adequately to anabolic agents such as fluoride or PTH.

IT **83139-29-1**, Human parathyroid hormone(1-34) amide
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(parathormone and antiresorptive agents anabolic effects at skeletal sites in osteopenia and estrogen depletion in ageing)

L18 ANSWER 24 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 1995:643176 HCAPLUS
DN 123:25845
TI Solution Structure and Adenylyl Cyclase Stimulating Activities of C-Terminal Truncated Human Parathyroid Hormone Analogs
AU Neugebauer, W.; Barbier, J.-R.; Sung, W. L.; Whitfield, J. F.; Willick, G. E.
CS Institute for Biological Sciences, National Research Council of Canada, Ottawa, ON, K1A 0R6, Can.
SO Biochemistry (1995), 34(27), 8835-42
CODEN: BICHAW; ISSN: 0006-2960
PB American Chemical Society
DT Journal
LA English
AB Analogs of human parathyroid hormone (hPTH) truncated at the C-terminal end have been studied for adenylyl cyclase (AC) activity and for solution conformation by CD spectroscopy. Analogs of hPTH-(1-34)-NH2, containing the first 28-31 residues, had only a slightly diminished ability to stimulate AC in rat osteosarcoma (ROS) cells as compared to that of the parent analog. CD data on hPTH-(16-34)-NH2 and C-terminal deletion mutants of hPTH-(1-34)-NH2 supported the presence of a partially stable α-helix over residues 17-28. A carboxyl-terminal mutant, hPTH-(1-30)-OH, showed both reduced helix and greatly reduced AC-stimulating activity as compared to the corresponding amide analog. In contrast, both of these analogs, in the presence of palmitoyloleoylphosphatidylserine (POPS) vesicles, showed an equal stabilization of α-helix. All other analogs showed at least some enhancement of α-helix in the presence of POPS. However, both in neutral, aqueous buffer and in POPS, the relative amount of α-helix decreased greatly as the peptide was shortened below the 1-28 sequence. These data provide addnl. support for an amphiphilic α-helix over residues 21-28 being the conformation for receptor binding of hPTH for stimulation of AC activity. Modeling human

parathyroid hormone-related peptide as an α-helix over this same region, and comparison to hPTH, suggests that both may bind via the hydrophobic face to the receptor.

IT **83139-29-1**, HPTH-(1-34)-NH2
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(solution structure and adenylyl cyclase stimulating activities of C-terminal truncated human parathyroid hormone analogs)

L18 ANSWER 25 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 1995:624462 HCAPLUS
DN 123:286708
TI A versatile method for the synthesis of peptide (alkyl)amides using recombinant and solid-phase procedures
AU Fukuda, Tsunehiko; Nakagawa, Shizue; Tamakashi, Yuri; Hamana, Takumi; Nakamura, Junko; Kawase, Masahiro; Taketomi, Shigehisa; Ishibashi, Yoshihiro; Nishimura, Osamu
CS Pharmaceutical Research Laboratories II, Takeda Chemical Industries, Ltd., Osaka, 532, Japan
SO Peptide Chemistry (1995), Volume Date 1994, 32nd, 13-16
CODEN: PECHDP; ISSN: 0388-3698
DT Journal
LA English
AB The simultaneous chemical cleavage and amidation of recombinant fusion proteins, and of smaller peptides is described. This reaction is widely applicable, highly side-specific, and gives peptidyl amides and alkylamides in good yields in a simple procedure. Thus, site specific cyanation of a mutant human parathyroid hormone [hPTH(1-84)] fusion protein, [Cys35]-hPTH(1-84), with 1-cyano-4-(dimethylamino)pyridinium tetrafluoroborate (I) gave the cyanated protein [Cys(CN)35]-hPTH(1-84) in nearly quant. yield. Treatment of the cyanated protein with aqueous ammonia or alkylamines RNH2 [R = Me, Et, (CH2)5Me, CH2CH2Ph] resulted in simultaneous cleavage and amidation to give peptide amides hPTH(1-34)NHR in 9-62% yields. Similarly, treatment of pGlu-His-Trp-Ser-Tyr-D-Leu-Leu-Arg-Pro-Cys-Tyr-OH with I, followed by reaction with EtNH2, gave LH-RH analog pGlu-His-Trp-Ser-Tyr-D-Leu-Leu-Arg-Pro-NHEt in 32% yield.

IT **83139-29-1P**, HPTH-(1-34)-NH2
RL: SPN (Synthetic preparation); PREP (Preparation)
(a versatile method for the synthesis of peptide (alkyl)amides using recombinant and solid-phase procedures)

L18 ANSWER 26 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 1995:217380 HCAPLUS
DN 122:1241
TI Stabilized NMR structure of the hypercalcemia of malignancy peptide PTHrP[Ala-26](1-34)amide
AU Barden, Julian A.; Kemp, Bruce E.
CS Department of Anatomy and Histology, University of Sydney, Sydney, NSW, 2006, Australia
SO Biochimica et Biophysica Acta, Protein Structure and Molecular Enzymology (1994), 1208(2), 256-62
CODEN: BBAEDZ; ISSN: 0167-4838
PB Elsevier B.V.
DT Journal
LA English
AB The structure of the biol. active mutant PTHrP[Ala-26](1-34)amide in 10% trifluoroethanol was studied by two-dimensional proton NMR spectroscopy. Complete assignments of all backbone and side chain hydrogens were made with the aid of totally correlated and nuclear Overhauser effect spectroscopy. The NMR data were utilized in the distance geometry

algorithm (DIANA) and the resulting family of structures further refined using dynamic simulated annealing (X-PLOR). The major structural features include two segments of α-helix extending from Glu-4 to Lys-13 and from Phe-21/Phe-22 to Ala-34, with a turn from Gln-16 to Arg-19 and a hinge around Ser-14/Ile-15. A close resemblance to the structure of PTH(1-34)amide in water was noted. A comparison of the structural features common to PTH and PTHrP in different solvents was made which enabled the key structural features likely to be involved in PTH receptor binding to be identified.

IT **83139-29-1**
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(parathormone-related peptide mutant conformation and parathormone receptor binding activity)

L18 ANSWER 27 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 1995:16541 HCAPLUS
DN 122:188102
TI Chemical cleavage of recombinant fusion proteins to yield peptide amides
AU Nakagawa, Shizue; Tamakashi, Yuri; Hamana, Takumi; Kawase, Masahiro; Taketomi, Shigehisa; Ishibashi, Yoshihiro; Nishimura, Osamu; Fukuda, Tsunehiko
CS Pharmaceutical Research Laboratories II, Takeda Chemical Industries Ltd., Osaka, 532, Japan
SO Journal of the American Chemical Society (1994), 116(12), 5513-14
CODEN: JACSAT; ISSN: 0002-7863
DT Journal
LA English
OS CASREACT 122:188102
AB A versatile chemical method for synthesis of peptides with a C-terminal amide or alkyl-amide structure from a recombinant product is described. The method comprises the cyanation of the cysteine residue which links the objective peptide and fused protein with 1-cyano-4-dimethylaminopyridinium fluoroborate followed by treatment with ammonia or appropriate alkylamines. The reaction mixts. gave very simple HPLC profiles, and the peptidyl-amides produced were easily purified.
IT **83139-29-1P**, Human parathyroid hormone(1-34) amide
RL: SPN (Synthetic preparation); PREP (Preparation)
(preparation of, via cyanation-aminolysis of cysteine-containing fusion protein)

L18 ANSWER 28 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 1993:73779 HCAPLUS
DN 118:73779
TI NMR solution structure of the [Ala26]parathyroid-hormone-related protein(1-34) expressed in humoral hypercalcemia of malignancy
AU Ray, Fiona R.; Barden, Julian A.; Kemp, Bruce E.
CS Dep. Anat., Univ. Sydney, 2006, Australia
SO European Journal of Biochemistry (1993), 211(1-2), 205-11
CODEN: EJBCAI; ISSN: 0014-2956
DT Journal
LA English
AB The structure of the biol. active N-terminal domain of human parathyroid-hormone-related protein (residues 1-34) containing an Ala substituted for a His in position 26 was studied by 2-dimensional proton NMR spectroscopy. Unambiguous NMR assignments of all backbone and side-chain hydrogens were made with the aid of totally correlated spectroscopy expts., which provided through-bond 1H-1H connectivities, and NOE spectroscopy, which provided through-space and sequential backbone connectivities. The NMR data were utilized in distance-geometry

algorithms to generate a family of structures. The major structural features include 2 segments of α-helix extending from Glu4 to Lys13 and from Leu27 to Thr33, with 2 turns from Gln16 to Arg19 and Phe22 to His25. A salt-bridge appears likely between Arg20 and Glu30 which may be critical for holding the receptor-binding domain together.

IT **83139-29-1**
RL: BIOL (Biological study)
(cAMP formation response to, in osteogenic sarcoma cells, structure in relation to)

L18 ANSWER 29 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 1989:401577 HCAPLUS
DN 111:1577
TI Parathyroid hormone antagonists with simplified synthetic methodology and their use
IN Rosenblatt, Michael; Caporale, Lynn H.; Nutt, Ruth F.; Levy, Jay J.; Chorev, Michael
PA Merck and Co., Inc., USA
SO U.S., 5 pp.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 4771124	A	19880913	US 1987-54359	19870526
	EP 293159	A2	19881130	EP 1988-304671	19880523
	EP 293159	A3	19900509		
	R: AT, BE, CH, DE, ES, FR, GB, IT, LI, LU, NL, SE				
	DK 8802854	A	19890213	DK 1988-2854	19880525
	JP 63316800	A2	19881226	JP 1988-127207	19880526
PRAI	US 1987-54359	A	19870526		

AB The use of peptide hormone analog as inhibitors of their resp. naturally occurring peptide hormones and methods of synthesis of such analogs are described. The structure of the peptide hormone analog is exemplified by parathyroid hormone (PTH) wherein Trp23 is substituted by L-Phe or other hydrophobic amino acids such as Leu, Nle, Val, Tyr, β-naphthylalanine and α-naphthylalanine. The analogs were prepared by a modification of Merrifield's solid-phase method using 4-methylbenzhydrylamine HCl resin, lyophilized, and purified by reversed-phase HPLC on Vydac C4 bonded silica with aqueous CH3CN gradient and 0.1% CF3CO2H. [Nle8,18,125I-Tyr34] bovine PTH (1-34)NH2 was used in a receptor binding assay with bovine renal cortical plasma membranes. High specific binding (85%) was consistently obtained.

IT **83139-29-1**
RL: BIOL (Biological study)
(antagonist analogs of)

L18 ANSWER 30 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 1988:637022 HCAPLUS
DN 109:237022
TI Pharmaceuticals containing parathormone for the treatment of cataract
IN Hori, Masayuki; Uzawa, Toyonobu; Noda, Toshiharu
PA Toyo Jozo Co., Ltd., Japan
SO Jpn. Kokai Tokkyo Koho, 5 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

PI JP 63060940 A2 19880317 JP 1986-205704 19860901
PRAI JP 1986-205704 19860901
AB Cataract is treated or prevented by parathormone. After human parathormone (1-34) (h-PTH) (6 μg/kg) was given to galactose-rich fed rats for 13 days, cataract was observed in 3 out of 12 rats, vs 8 out of 12 rats without h-PTH treatment. Injection solution was formulated containing 60 mg
h-PTH, 10 g mannitol, and 2L H2O.
IT **83139-29-1**
RL: BIOL (Biological study)
(cataract treatment with)

L18 ANSWER 31 OF 31 HCAPLUS COPYRIGHT 2006 ACS on STN
AN 1982:582837 HCAPLUS
DN 97:182837
TI Studies on peptides. CX. Solution synthesis of the tetratriacontapeptide amide corresponding to positions 1 to 34 of human parathyroid hormone
AU Funakoshi, Susumu; Yajima, Haruaki; Shigeno, Chohei; Yamamoto, Itsuo; Morita, Rikushi; Torizuka, Kanji
CS Fac. Pharm. Sci., Kyoto Univ., Kyoto, 606, Japan
SO Chemical & Pharmaceutical Bulletin (1982), 30(5), 1738-46
CODEN: CPBTAL; ISSN: 0009-2363
DT Journal
LA English
GI

H-Ser-Val-Ser-Glu-Ile-Gln-Leu-Met-His-Asn-

Leu-Gly-Lys-His-Leu-Asn-Ser-Met-Glu-Arg-

Val-Glu-Trp-Leu-Arg-Lys-Lys-Leu-Gln-Asp-

Val-His-Asn-Phe-NH_2 I

AB The title peptide (I) was prepared by fragment condensations in solution The final protected peptide was deblocked by 1M CF3SO3H-thioanisole in CF3CO2H. Synthetic I exhibited an activity of 3380-4400 IU/mg when assayed by the mouse bone adenyl cyclase assay.
IT **83139-29-1P**
RL: SPN (Synthetic preparation); PREP (Preparation)
(preparation of, as tetratriacontapeptide amide of human parathyroid hormone)

=> fil uspatall
FILE 'USPATFULL' ENTERED AT 16:13:03 ON 23 MAR 2006
CA INDEXING COPYRIGHT (C) 2006 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'USPAT2' ENTERED AT 16:13:03 ON 23 MAR 2006
CA INDEXING COPYRIGHT (C) 2006 AMERICAN CHEMICAL SOCIETY (ACS)

=> d l19 bib abs hitrn tot

L19 ANSWER 1 OF 8 USPATFULL on STN
AN 2005:293498 USPATFULL
TI Method for fostering bone formation and preservation
IN Vignery, Agnes, Branford, CT, UNITED STATES

Mehta, Nozer M., Randolph, NJ, UNITED STATES
Gilligan, James P., Union, NJ, UNITED STATES
PI US 2005256047 A1 20051117
AI US 2005-128095 A1 20050511 (11)
PRAI US 2004-571200P 20040514 (60)
DT Utility
FS APPLICATION
LREP OSTROLENK FABER GERB & SOFFEN, 1180 AVENUE OF THE AMERICAS, NEW YORK, NY, 100368403, US
CLMN Number of Claims: 45
ECL Exemplary Claim: 1
DRWN 8 Drawing Page(s)
LN.CNT 1081
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method of inducing bone formation in a subject in need of such inducement comprises the steps of mechanically inducing an increase in osteoblast activity in the subject and elevating blood concentration of at least one bone anabolic agent in the subject. The method steps may be performed in any order, but in sufficient time proximity that the elevated concentration of the anabolic agent and the mechanically induced increase in osteoblast activity overlaps. The method may additionally comprise providing the subject with an elevated blood concentration of at least one antiresorptive agent, wherein the elevated concentration is sufficient to prevent resorption of new bone growth produced due to the osteoblast activity. Use of the method permits targeting of specific bones of the subject for bone production and preservation, faster bone production and earlier discontinuation of bone anabolic pharmaceuticals. Kits adapted for performing the method are provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
IT **83139-29-1**
(method for fostering bone formation and preservation using anabolic agent to increase osteoblast activity and antiresorptive agent)

L19 ANSWER 2 OF 8 USPATFULL on STN
AN 2004:254298 USPATFULL
TI Method for selective radioactive marking of peptides
IN Koglin, Norman, Leipzig, GERMANY, FEDERAL REPUBLIC OF
Lang, Manja, Leipzig, GERMANY, FEDERAL REPUBLIC OF
Beck-Sickinger, Annette G., Leipzig, GERMANY, FEDERAL REPUBLIC OF
PI US 2004197824 A1 20041007
AI US 2003-653779 A1 20030902 (10)
PRAI US 2002-407179P 20020830 (60)
DT Utility
FS APPLICATION
LREP Horst M. Kasper, 13 Forest Drive, Warren, NJ, 07059
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN 13 Drawing Page(s)
LN.CNT 867
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to a method for selective radio marking of peptides. It is the object to present a generally applicable method for the production of radio ligands. The object is accomplished by the use of photo labile protective groups in peptide synthesis, wherein the photo labile groups block pre-given amino groups for such time until the desired derivative formation of a specific amino group is performed with a .sup.3H-containing group and the protective groups are split off by UV radiation.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
IT **83139-29-1**
(preparation of selectively labeled peptides using solid-phase peptide synthesis and orthogonal protection schemes)

L19 ANSWER 3 OF 8 USPATFULL on STN
AN 2004:31740 USPATFULL
TI PTH derivatives resistant to skin proteases
IN Peri, Krishna G., Montreal, CANADA
High, Kim, St-Lazare, CANADA
Bergeron, Annie, Repentigny, CANADA
Moffett, Serge, St-Caurent, CANADA
Abribat, Thierry, Montreal, CANADA
PI US 2004023882 A1 20040205
AI US 2003-440473 A1 20030516 (10)
PRAI US 2002-378072P 20020516 (60)
DT Utility
FS APPLICATION
LREP Michael R. Krawzsenek, FULBRIGHT & JAWORSKI L.L.P., Suite 2400, 600 Congress Avenue, Austin, TX, 78701
CLMN Number of Claims: 26
ECL Exemplary Claim: 1
DRWN 3 Drawing Page(s)
LN.CNT 717
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Protease-resistant analogues of biologically active derivatives of human PTH are described. These analogs are intended for use in therapeutic preparations for the treatment of various medical conditions in which bone loss is encountered or is susceptible of being encountered. The analogs specified are hPTH(1-34) and hPTH(1-31). More particularly, protease-resistant analogs of PTH adapted for transdermal administration are described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
IT **83139-29-1P**
(synthesis and therapeutic uses of PTH derivs. resistant to skin proteases)

L19 ANSWER 4 OF 8 USPATFULL on STN
AN 2003:89373 USPATFULL
TI Parathyroid hormone analogues for the treatment of osteoporosis
IN Barbier, Jean-Rene, Gatineau, CANADA
Morley, Paul, Ottawa, CANADA
Neugebauer, Witold, Ottawa, CANADA
Whitfield, James F., Ottawa, CANADA
Willick, Gordon E., Orleans, CANADA
PA National Research Council of Canada, Ontario, CANADA (non-U.S. corporation)
PI US 6541450 B1 20030401
AI US 2000-536785 20000328 (9)
RLI Division of Ser. No. US 1997-904760, filed on 1 Aug 1997, now patented, Pat. No. US 6110892 Continuation-in-part of Ser. No. US 1996-691647, filed on 2 Aug 1996, now patented, Pat. No. US 5955425 Continuation-in-part of Ser. No. US 1994-262495, filed on 20 Jun 1994, now patented, Pat. No. US 5556940 Continuation-in-part of Ser. No. US 940760
PRAI US 1997-40560P 19970314 (60)
DT Utility
FS GRANTED

EXNAM Primary Examiner: Russel, Jeffrey E.
LREP Nixon & Vanderhye
CLMN Number of Claims: 18
ECL Exemplary Claim: 5
DRWN 28 Drawing Figure(s); 12 Drawing Page(s)
LN.CNT 1247
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention describes analogues of human parathyroid hormone which have increased activities in bone restoration, and increased bioavailability. The peptides described are derivatives of hPTH-(1-31) which are cyclized for example, by formation of lactams between either Glu.sup.22 and Lys.sup.26 or Lys.sup.26 and Asp.sup.30. In addition, the natural Lys.sup.27 may be substituted by either a Leu or other hydrophobic residues, such as Ile, norleucine, Met, Val, Ala, Trp, or Phe. Typically, these analogues have enhanced abilities to stimulate adenylyl cyclase in rat osteosarcoma cells, and show increased activities in bone restoration, using the ovariectomized rat model. The analogues also show enhanced activities and bioavailabilities, as demonstrated by their hypotensive effects in the rat. An assay which correlates hypotensive activity with osteogenic activity is also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
IT **83139-29-1P**
(preparation of parathyroid hormone analogs for treatment of osteoporosis)

L19 ANSWER 5 OF 8 USPATFULL on STN
AN 1998:48374 USPATFULL
TI Continuous low-dose administration of parathyroid hormone or its agonist
IN Chorev, Michael, Brookline, MA, United States
Rosenblatt, Michael, Newton Centre, MA, United States
PA Beth Israel Deaconess Medical Center, Boston, MA, United States (U.S. corporation)
PI US 5747456 19980505
AI US 1996-724539 19960930 (8)
RLI Continuation-in-part of Ser. No. US 1994-359293, filed on 19 Dec 1994, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Tsang, Cecilia J.; Assistant Examiner: Touzeau, P. Lynn
LREP McGowan, William, Conway, John D.Fish & Richardson
CLMN Number of Claims: 21
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 566
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method of promoting bone formation in a human patient, which includes the step of administering continuously to the patient parathyroid hormone or its agonist for a period of at least one month at a dosage between 10 and 400 units/24 hrs. Also disclosed are novel parathyroid hormone agonists.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
IT **83139-29-1P**, 1-34-Human PTH-NH2
(preparation and continuous low-dose administration of parathyroid hormone or its agonist)

L19 ANSWER 6 OF 8 USPATFULL on STN
AN 97:77886 USPATFULL

TI Monitoring method of renal lesions without clinical signs
IN Ogata, Etsuro, Tokyo, Japan
Kurokawa, Kiyoshi, Tokyo, Japan
Uchida, Shunya, Tokyo, Japan
Kishida, Akiko, Kanagawa, Japan
PA Asahi Kasei Kogyo Kabushiki Kaisha, Osaka, Japan (non-U.S. corporation)
PI US 5662604 19970902
AI US 1995-495635 19950919 (8)
WO 1993-JP178 19930212
19950919 PCT 371 date
19950919 PCT 102(e) date
DT Utility
FS Granted
EXNAM Primary Examiner: Mendez, Manuel
LREP Young & Thompson
CLMN Number of Claims: 8
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 503
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A screening method for detecting renal lesions in diabetic patients not yet exhibiting symptoms of nephropathy, comprising administering a predetermined dose of parathyroid hormone, analogue thereof or derivative thereof to the patients with an albumin excretion rate (AER) of microalbumin being below 15 μg/min., measuring urinary excreted N-acetyl-β-D-glucosaminidase (NAG) and urinary creatinine in urinary samples taken 60±10 minutes before and after administration of PTH, and monitoring the increased ratio of urinary excreted NAG (U/g Cr) in post-administration of PTH to the urinary excreted NAG (U/g Cr) in pre-administration of PTH for a value less than 2.3. The screening method provides the possibility of detecting renal lesions without clinical signs of diabetic nephropathy, using a preparation essentially consisting of active ingredient of PTH.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
IT **83139-29-1**, Human PTH (1-34)-NH2
(post PTH urinary N-acetyl-β-D-glucosaminidase in diagnosis of nonsymptomatic diabetic nephropathy)

L19 ANSWER 7 OF 8 USPATFULL on STN
AN 96:85218 USPATFULL
TI Parathyroid hormone analogues for the treatment of osteoporosis
IN Willick, Gordon E., Orleans, Canada
Whitfield, James F., Ottawa, Canada
Surewicz, Witold, Orleans, Canada
Sung, Wing L., Gloucester, Canada
Neugebauer, Witold, Ottawa, Canada
PA National Research Council of Canada, Ottawa, Canada (non-U.S. corporation)
PI US 5556940 19960917
AI US 1994-262495 19940620 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Russel, Jeffrey E.
CLMN Number of Claims: 2
ECL Exemplary Claim: 2
DRWN 11 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 401
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Certain analogues of human parathyroid hormone (hPTH) have been found to

be effective for the treatment of osteoporosis, while showing decreased side effects. Analogues showing this effect include all sequences from hPTH-(1-28)-NH.sub.2 to hPTH-(1-31)-NH.sub.2 and all sequences from [Leu.sup.27]-hPTH-(1-28)-NH.sub.2 to [Leu.sup.27]-hPTH-(1-33)-NH.sub.2. Also included are cyclic analogues cyclo(Lys.sup.26 -Asp.sup.30) [Leu.sup.27]-hPTH-(1-34)NH.sub.2 and cyclo (Lys.sup.27 -Asp.sup.30)-hPTH-(1-34)-NH.sub.2. Analogues in the form of the carboxyl terminal amide are particularly effective.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
IT **83139-29-1P**
(preparation of parathyroid hormone analogs for treatment of osteoporosis)

L19 ANSWER 8 OF 8 USPATFULL on STN
AN 88:59158 USPATFULL
TI Parathyroid hormone antagonists with simplified synthetic methodology
IN Rosenblatt, Michael, Ardmore, PA, United States
Caporale, Lynn H., Lansdale, PA, United States
Nutt, Ruth F., Green Lane, PA, United States
Levy, Jay J., Paoli, PA, United States
Chorev, Michael, Jerusalem, Israel
PA Merck & Co., Inc., Rahway, NJ, United States (U.S. corporation)
PI US 4771124 19880913
AI US 1987-54359 19870526 (7)
DT Utility
FS Granted
EXNAM Primary Examiner: Phillips, Delbert R.
LREP Caruso, Charles M., Pfeiffer, Hesna J.
CLMN Number of Claims: 3
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 348
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to the use of peptide hormone analogues as inhibitors of their respective naturally occurring peptide hormone and methods of synthesis of such analogues. The structure of the peptide hormone analogues is exemplified by parathyroid hormone wherein Trp.sup.23 is substituted by L-Phe or other hydrophobic amino acids such as Leu, Nle, Val, Tyr, beta-napthylalanine and alpha-napthylalanine.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
IT **83139-29-1**
(antagonist analogs of)

=> d his

(FILE 'HOME' ENTERED AT 16:04:07 ON 23 MAR 2006)
SET COST OFF

FILE 'HCAPLUS' ENTERED AT 16:04:25 ON 23 MAR 2006
L1 1 S US20040197323/PN OR (US2004-761481# OR WO2004-US01633 OR US20
E MEHTA N/AU
L2 52 S E3,E12
E MEHTA NOZER/AU
L3 28 S E2-E4
E STERN W/AU
L4 125 S E3-E9,E22-E24
L5 11 S E24-E28
E GILLIGAN J/AU

```
L6            57 S E3,E8,E10-E12
                 E UNIGENE/PA,CS
L7           175 S UNIGEN?/PA,CS
                 SEL RN L1

     FILE 'REGISTRY' ENTERED AT 16:07:35 ON 23 MAR 2006
L8            34 S E1-E34
L9             1 S L8 AND 34/SQL
L10          277 S SVSEIQLMHNLGKHLNSMERVEWLRKKLQDVHNF/SQSP
L11           61 S L10 AND 34/SQL
L12           60 S L11 NOT L9
L13            2 S L12 AND PARATH?
L14            1 S L13 AND 83139-29-1
                 SAV L14 RUSSEL761/A

     FILE 'HCAOLD' ENTERED AT 16:10:51 ON 23 MAR 2006
L15            0 S L14

     FILE 'HCAPLUS' ENTERED AT 16:11:01 ON 23 MAR 2006
L16           31 S L14
L17            1 S L16 AND L1-L7
L18           31 S L16,L17

     FILE 'USPATFULL, USPAT2' ENTERED AT 16:11:33 ON 23 MAR 2006
L19            8 S L14

     FILE 'REGISTRY' ENTERED AT 16:12:26 ON 23 MAR 2006

     FILE 'HCAPLUS' ENTERED AT 16:12:34 ON 23 MAR 2006

     FILE 'USPATFULL, USPAT2' ENTERED AT 16:13:03 ON 23 MAR 2006

=>
```